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XXIII.

A STUDY OF THE DEVELOPMENT OF THE NOSE AND ITS ACCESSORY CAVITIES.*

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This paper must be considered as only a preliminary one, for the field is so broad that the author has been unable to cover it exhaustively.

The primary origin of the olfactory organ is not well understood: possibly it may have arisen by a modification of primitive integumentary sense organs.

In its simplest form the olfactory organ consists of two pit-like depressions of the integument of the snout opening on the surface by external nostrils. It is lined by an epithelium which is connected with the brain by the olfactory nerves. The olfactory organs of all the true fishes consist of sack-like depressions which do not communicate with the mouth. In the course of development each external nostril is divided by a fold of skin into two openings, thus forming an anterior and posterior nostril.

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Dipnoi are fishes which resemble amphibia in several ways. They live in shallow streams and bury themselves in the mud during the dry season. The air bladder is converted into a funtional lung and the nasal cavities communicate with the mouth as well as with the exterior. In consequence of this, anterior or external, and posterior or internal nostrils (choanae) can be distinguished and a free passage is thus formed through which air passes and the olfactory organ takes on an important relation to the respiratory apparatus.

In amphibia the structure of the olfactory organ becomes modified in correspondence with the change in the mode of respiration. The nasal chambers become differentiated into an olfactory and respiratory portion and an extension of the olfactory surface takes place by the formation of one or more prominences on the floor and side walls of the nasal cavities. These prominences correspond to the turbinals of higher forms

Glands, situated under the olfactory mucous membrane, can now be found, and the secretion from these glands moistens the mucous membrane of the nasal cavity.

In reptiles, owing to the growth of the brain and facial region and the formation of a secondary palate, the olfactory organs from reptiles onward gradually come to be situated more ventrally beneath the cranium.

The secondary palate is formed by the pterygopalatine arch and is well developed in all reptiles. In snakes and lizards this arch is more or less movable and free from the base of the skull, but in crocodiles it meets with its fellow in the median line, and the shelf-like palatine processes of the superior maxillary bones unite with the palatines. Thus a secondary roof is formed to the mouth, distinct from the true (sphenoidal) base of the skull. The cavity thus formed closes in the posterior prolongation of the nasal chambers which consequently becomes sharply differentiated from the mouth and the posterior nares open farther back into the pharynx or nasopharynx instead of the mouth.

In birds there is an anterior nasal chamber, lined by stratified epithelium, and an olfactory chamber situated above and posterior to the former. The olfactory chamber contains the ethmoidal turbinal, the form of which varies greatly. It

may be simply a prominence or more or less rolled upon itself.

Corresponding to the much more marked development of the facial portion of the skull, the nasal cavity of mammals is much larger and there is a more extensive development of the turbinals.

The turbinals become very complicated and form a sponge-like labyrinth, the cell-like compartments of which are lined throughout with mucous membrane. The number of olfactory turbinals varies considerably, but in most mammals there are at least five. These olfactory turbinals are generally described as ethmoidal turbinals because they are usually supported by the ethmoid bone. The turbinal of lower vertebrates corresponds to the maxillary turbinal in mammals situated in the lower or anterior part of each nasal chamber and supported by the superior maxillary bone.

The maxillary turbinal in mammals no longer possesses an olfactory epithelium and in microsmatic mammals (seals, whales, monkeys and man), the lower portion of the ethmoidal turbinal also loses its olfactory epithelium. This change in the mucous membrane of this portion of the nose corresponds to the change in function, for its principal function now is to clean, warm and moisten the inspired air.

The author wishes here to acknowledge his indebtedness to Dr. Robert Wiedersheim's book "Comparative Anatomy of Vertebrates," from which the statements above have been compiled. With this description of the development of the olfactory organ as a guide, let us study some members of the different classes of vertebrates more carefully.

FISH. The olfactory organ in the Lake Erie white fish is composed of two symmetrical cavities situated in the anterior part of the head and separated from each other by a thick septum.

Each cavity is somewhat hemispherical in shape and communicates with the exterior by two small round openings in the roof of the cavity. These openings are situated one behind the other and so may be designated as anterior and posterior nostrils.

The external wall and floor of each cavity is smooth, but the median wall (septum) supports a rounded prominence,

from which radiating processes extend into the nasal cavity (Plate I, 3). These processess correspond to the turbinals in higher vertebrates. The two nostrils open just above these processes and thus allow a free circulation of water through the cavity (Plate I, 1 and 2).

The nasal cavity is separated from the mouth by a rather thick wall, but in the posterior inferior part of the cavity there is a slight depression extending downward toward the mouth (Plate I, 5).

TURTLE. The olfactory organ in the turtle consists of two symmetrical cavities separated from each other by a thick septum. The respiratory part of each nasal fossa is a tube-like structure extending doward and backward and opening into the anterior part of the mouth (Plate II., Figure A, 1 and 4). The olfactory region is a spherical chamber situated above the respiratory region with which it communicates by an oval opening in its floor. (Plate II. and IV., Fig. A and D, 3). Projecting into each fossa, from the lateral walls, there are two prominences (Plate II. and IV., Fig. A and D, 11) which correspond to the turbinals in higher forms. These prominences form the anterior and inferior boundaries of the olfactory region. The anterior prominence extends downward across the nasal fossa and unites with the septum, thus partially subdividing the respiratory region into an anterior and posterior portion.

DUCK. The nasal fossae in the duck are two symmetrical cavities which communicate with each other at their anterior and posterior extremities, the septum being incomplete at these points.

The maxillary turbinal is shaped like an inverted T, attached to the roof of the nasal chamber and is suggestive of the turbinal with a double coil seen in higher forms of vertebrates (Plate V. and VI., Fig. A, B and C, 1).

There are two ethmoidal turbinals. The anterior inferior one (Plate V. and VI., Fig. A. E. F. and G. 2) is a double coil of which the superior coil curves upward describing nearly a complete circle; the inferior coil is scroll-shaped making two turns within itself (Plate V. and VI., Fig. G, 2).

The superior ethmoidal turbinal is a simple structure (Plate III, Figures A and G, 3), consisting of a rounded prominence from which two irregular processes extend downward into the nasal fossa.

There are no accessory sinuses of the nose but in addition to the numerous small air cells the greater part of each superior maxillary bone is occupied by a large air cell (Plate V. and VI, Figures F and G, 10). These air cells, like the

other pneumatic cells in the bird's skeleton, make the bones lighter in weight.

Dog. The dog's nose is a very complicated structure. The maxillary turbinal is attached to the superior maxillary bone by a single shelf-like process which is much broader at its posterior than at its anterior end. From this process, numerous curved branches are given off, which form an intricate labyrinth almost completely filling the anterior part of each nasal fossa. (Plates VII, VIII, IX and X, Fig. A, B, C, D, and E, 1).

The ethmoidal turbinal is an exceedingly complex structure. It consists of a mass of thin delicate scrolls of which at least five radiate from the convex surface of the cribiform plate. It articulates with and is supported by the sphenoidal, frontal, nasal and superior maxillary bones and *some of its numerous branches extend into all the accessory cavities of the nose.* (Plates VI. to X. Fig. A, B, C, D, E, F, G and H, 2). One scroll extends forward over the maxillary turbinal and anteriorly it is supported by *a process which is attached to the external lateral wall of the maxillary sinus.* (Plates VII. to X., Fig. B, C, D, F and G, 12).

The ethmoidal cells are cavities in the scrolls of the ethmoidal turbinals or cavities between the scrolls and the surrounding bones of the skull. (Plates VII, VIII, IX and X, Fig. B, C, D, H, J and K, 4).

The maxillary sinus is hollowed out of the superior maxillary bone and is only partially separated from the nasal fossa by the ethmoid bone. (Plates VI to X, Figures B, C, F and G, 11). The ostium maxillare is large and extends downward to the floor of the cavity, opening into the nasal fossa in the inferior meatus just posterior to the maxillary turbinal. (Plate VI and IX, Fig. A, C and D, 3). The hiatus semilunaris is a groove formed in one of the processes of the ethmoidal turbinal and leads directly from the frontal and ethmoidal cells into the maxillary sinus so that these cells all drain into the maxillary sinus and thence into the inferior meatus (Plates VII and IX, Figures C and F, 14). The frontal sinus is situated between the two plates of the frontal bone and *its anterior inferior part is occupied by a double coil of the ethmoidal turbinal which is attached to the external wall of the sinus* (Plates VII,VIII, X. and XI, Fig. A, B, C, D, H, I and K, 5 and 6).

The sphenoidal sinuses (two in number) are separated from each other by a rather thick septum in the median line and *are nearly filled by scrolls of the ethmoidal turbinal* (Plate XI, Fig. I and K, 8).

The posterior part of the vomer is expanded into two lateral wings which unite with the ethmoidal bone and thus divide the posterior part of each nasal fossa into two superimposed cavities (Plates VII to X, Figures D, F, G and H, 18). The superior cavity contains most of the ethmoidal turbinal and is distinctly an olfactory chamber. The inferior cavity is simply a tube-like structure forming a direct communication between the anterior half of the nasal fossa and the nasopharynx, (Plates VII to X, Figures A, D, F and G, 21).

The function of the accessory cavities of the nose in the dog, seems to be to provide space for the tremendous development of the olfactory turbinals, to conduct air over the external surface of these structures and to furnish a system of drainage for them.

MONKEY. The olfactory organ in monkeys varies considerably in different species, but there are certain characteristics which are constant.

The maxillary turbinal is a comparatively simple structure and consists of a single or double coil. The superior coil is usually smaller than the inferior coil and is frequently only rudimentary or absent entirely. The turbinal is supported by the superior maxillary bone and its base forms a large part of the internal wall of the maxillary sinus.

The ethmoidal turbinals vary in number from two to five and are situated above the maxillary turbinal. They are also comparatively simple structures. *In some species they extend into the posterior superior part of the maxillary sinus.* The maxillary sinus is always present and opens into the middle meatus. *On its outer wall there is a bony ridge*, in some species slight, in others very prominent, *which partially subdivides the sinus.* (Plate XIII, 7.) This ridge is, I think, a rudiment of the ethmoid turbinal and probably accounts for the bony partition frequently seen in the maxillary sinus of man.

The ethmoidal cells vary greatly in size and number. In some species there are none, in some a single group opening into the middle or superior meatus, according to their location, and in others two groups, the anterior cells opening into the middle meatus and the posterior cells into the superior meatus. (Plate XII, 3.)

There are no frontal sinuses in most of the monkeys' skulls which I have examined but the frontal bone in this region is composed of cancellous tissue, and in some of the higher orders of monkeys the most anterior ethmoidal cells extend up between the two plates of the frontal bone. (Plate

XII, 1.) In the chimpanzee there is a well developed frontal sinus in each frontal bone with a thin bony septum in the median line. This sinus communicates directly with the anterior ethmoidal cell. (Plate XIII, 1.)

In those specimens in which there are no frontal sinuses and the ethmoidal cells are small or absent also, the orbits are situated very close together and this juxtaposition of the orbits may account for the disappearance of the frontal and ethmoidal sinuses, these sinuses having been crowded out by the orbits.

The sphenoidal sinuses vary greatly. In those monkeys in which the turbinal structures are simple and rudimentary, the sphenoidal sinuses are small or have disappeared entirely, but where the turbinals are less rudimentary, one or more sphenoidal sinuses are present in each lateral half of the sphenoidal bone and some show distinct evidences of turbinals extending into them, (Plates XII and XIII, 5 and 6.)

MAN. The general characteristics of the nose and its accessory sinuses in man are too well known to need a detailed description here. There are, however, certain structures which occur frequently enough to be considered fairly constant, and are of importance when studied in relation to the development of the nose. A third ethmoidal turbinal, and more rarely a fourth one, may be present. These structures are evidently rudiment of the more extensive development of the ethmoidal turbinal in lower forms and as is usual with such structures, they occur more frequently in the embryo than in the adult.

The maxillary sinus is sometimes partially divided into an anterior and posterior chamber by a bony partition extending across its floor (Plate XV., Figure B, 5), or up on its external lateral wall. Such partitions are, I think, rudiments of the ethmoidal turbinal which, in the dog, extends down into the maxillary sinus.

The frontal sinus sometimes communicates with an anterior ethmoidal cell (Plate XIV, Figure A 1), or the posterior inferior part of the sinus is encroached upon by the ethmoidal cells, or there are two sinuses in one lateral half of the skull, or the frontal sinus is absent.

The sphenoidal sinus sometimes communicates with a posterior ethmoidal cell or the sinus is divided into two sinuses, one of which opens directly into the nasal fossa and the other into an ethmoidal cell or both open into the superior meatus. (Plate XVII, Figure D. 6 and 7.) These variations are all easily explainable, I think, when they are compared with similar structures in lower vertebrates and are probably reversions to more primitive types.

The variations in the frontal sinuses correspond to the structure of these sinuses as seen in such animals as the dog and the monkey. The division of a sphenoidal sinus into two chambers is caused by a plate of the ethmoidal (or sphenoidal) turbinal extending across the sinus as it does in some monkeys.

Summarizing briefly, we find that the olfactory organ in fishes consists of two sack-like structures, each of which communicates with the exterior by two openings, but the nasal cavities do not communicate with the mouth. The turbinals are represented by prominences on the median wall. In dipnoi the nasal cavities open into the mouth and the nose then becomes a part of the respiratory system.

In reptiles the nasal cavities are subdivided into olfactory and respiratory regions and the turbinal structures are more highly developed. The secondary palate separates the posterior nares from the mouth and the nasal fossae are thus prolonged and open farther back into the nasopharynx.

In birds, the turbinal structures are all more complicated and the ethmoidal turbinals especially show a still higher degree of development.

In macrosmatic mammals (animals which have an acute sense of smell), the turbinal structures are exceedingly complicated scrolls forming intricate labyrinths which occupy the greater part of the nasal cavities. The accessory cavities of the nose all contain olfactory turbinals and are in intimate relation with these structures.

In microsmatic mammals (animals which do not have an acute sense of smell), the turbinals all show evidences of degeneration or reversion, and are comparatively simple structures. The accessory cavities contain little or no true turbinal tissue and their functional activity is lost.

It seems probable, therefore, that in man the accessory cavities of the nose are rudimentary structures.

The foregoing statements are based upon the study of the nose in fishes, frogs, alligators, turtles, birds, dogs, cats, wolves, foxes, bears, beavers, seals, rabbits, guinea pigs, lions, deer, sheep, cows, pigs, buffaloes, horses, elephants, monkeys and man.

I wish to thank Dr. Wm. T. Howard and Dr. L. W. Ladd for the facilities which they have given me for carrying on this work in their laboratories, Dr. R. G. Perkins and Dr. W. B. Chamberlin for doing the photographic work, Dr. C. A. Hamman and the Smithsonian Institute for loaning me material for study.

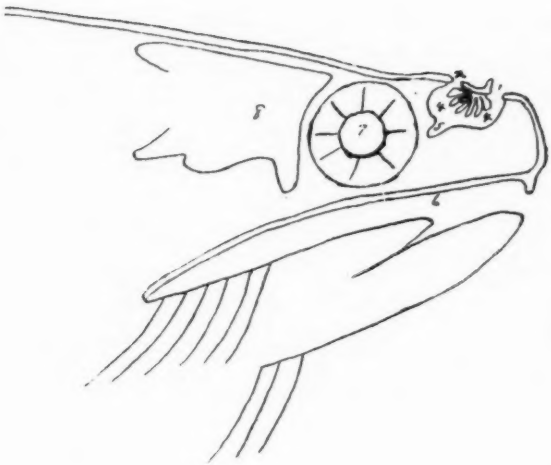

PLATE I.

Somewhat diagramatic drawing of a section through the nasal fossa of a fish.

1. Anterior naris.
2. Posterior naris (both of which open externally).
3. Turbinal.
4. Nasal fossa.
5. Prolongation of the nasal fossa downward toward the mouth.
6. Mouth.
7. Orbit.
8. Brain cavity.

PLATE II.

A. Longitudinal section through the left nasal fossa of a turtle.
1. Anterior naris.
2. Jacobson's organ.
3. Olfactory chamber.
4. Posterior naris.
5. Mouth.
6. Orbit.
7. Brain cavity.
8. Olfactory lobe of brain and olfactory nerves.
9. Septum.
10. Partition between nasal fossae and Jacobson's organ (Figure C). In the right fossa this partition has been cut away.
11. Turbinals.

PLATE III.

B. Transverse section through both nasal fossae of a turtle, looking anteriorly, 1—2—9, same as on Plate II.

E. Transverse section through the posterior part of both nasal fossae of a turtle, looking posteriorly, 4—5—6—8, same as on Plate II.

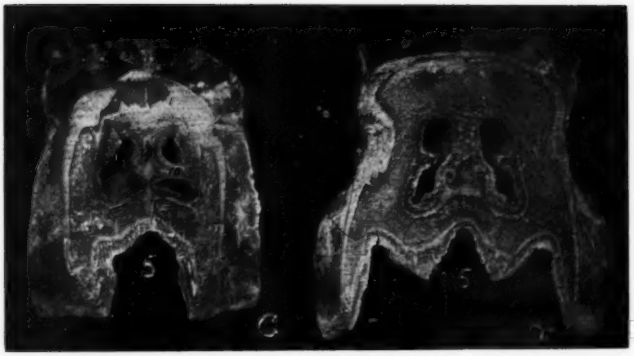

PLATE IV.

C & D. Transverse sections through both nasal fossae of a turtle, looking posteriorly, 3—5—9—10—11, same as on Plate II.

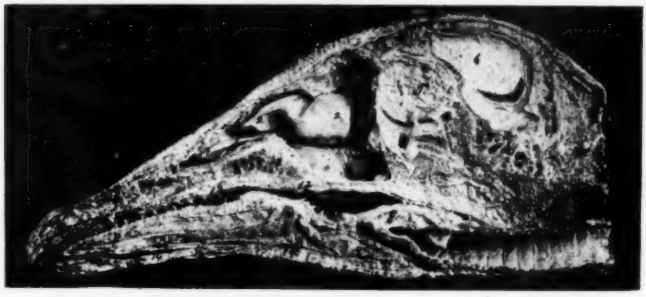

PLATE V.

A. Longitudinal section through the right nasal fossa of a duck.

1. Maxillary turbinal.
2. Inferior ethmoidal turbinal.
3. Superior ethmoidal turbinal.
4. Posterior naris.
5. Eye.
6. Brain.
7. Tongue.
8. Larynx.
9. Trachea.
10. Air cells in the bones.
11. Septum.
12. Roof of mouth.
13. Mouth.
14. Inferior maxilla.

PLATE VI.

Transverse section through both nasal fossae of a duck; B looking anteriorly; C D E F and G looking posteriorly. In B C and D the inferior maxilla is not shown, 1 to 14, as in Plate V.

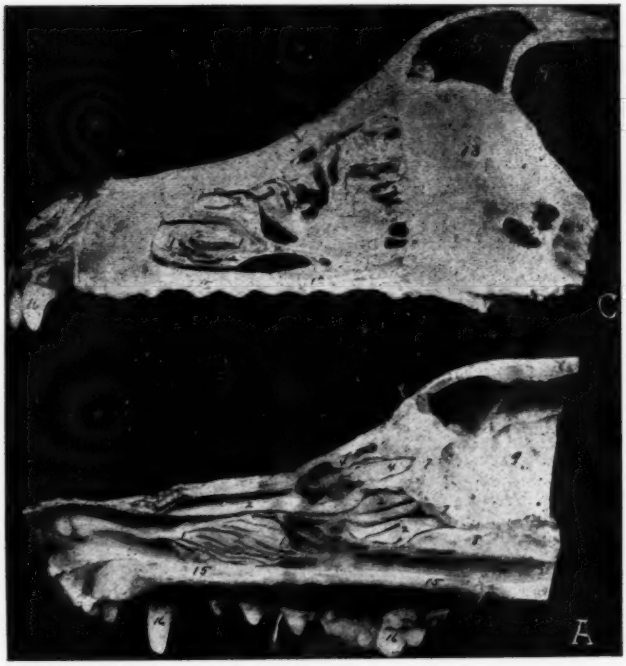

PLATE VII.

A. The right nasal fossa of a dog.
C. Section through the external third of the left fossa, looking toward the median line.

1. Maxillary turbinal.
2. Ethmoidal turbinal.
3. Ostium maxillare.
4. Ethmoidal turbinal in ethmoidal cells.
5. Ethmoidal turbinal in frontal sinuses.
6. Frontal sinus.
7. Cribriform plate.
8. Ethmoidal turbinal in sphenoidal sinus.
9. Brain cavity.
10. Posterior nares.
11. Maxillary sinus.
12. Ethmoidal turbinal extending into the maxillary sinus and attached to its external lateral wall.
13. Orbit.
14. Hiatus semilunaris extending from the frontal and ethmoidal cells into the maxillary sinus.
15. Palate.
16. Teeth.
17. Vomer.
18. Lateral wings of vomer.
19. Septum.
20. Presphenoid.
21. Respiratory tract in the posterior inferior part of the nares.

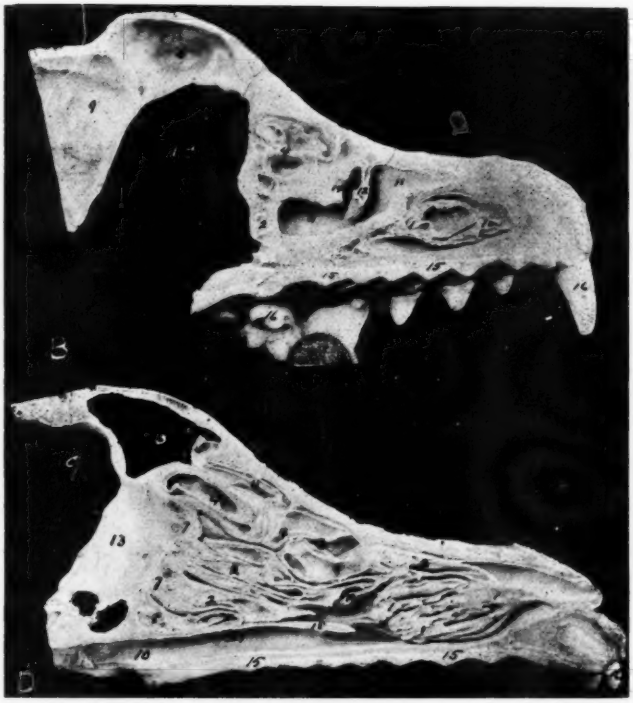

PLATE VIII.

B. Section showing the external third of the left nasal fossa of dog, looking toward the external wall.

D Section through the left nasal fossa, midway between the septum and the external wall, looking toward the latter, 1 to 21, as in Plate VII.

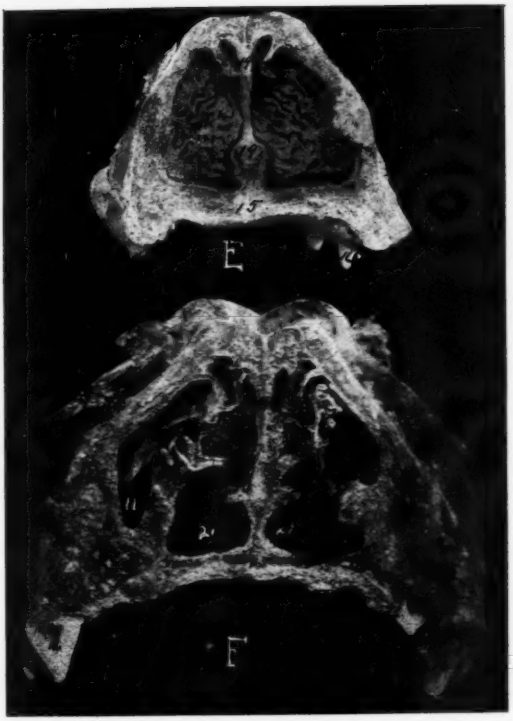

PLATE IX.

Transverse sections through both nasal fossa of the dog, cut slightly obliquely so as to pass through different parts of corresponding structures in each fossa, looking posteriorly.

1. Maxillary turbinal.
2. Ethmoidal turbinal.
3. Ostium maxillare.
4. Ethmoidal turbinal in ethmoidal cells.
5. Ethmoidal turbinal in frontal sinus.
6. Frontal sinus.
7. Cribriform plate.
8. Ethmoidal turbinal in sphenoidal sinus.
9. Brain cavity.
10. Posterior nares.
11. Maxillary sinus.
12. Ethmoidal turbinal extending into the maxillary sinus and attached to its external lateral wall.
13. Orbit.
14. Hiatus semilunaris extending from the frontal and ethmoidal cells into the maxillary sinus.
15. Palate.
16. Teeth.
17. Vomer.
18. Lateral wings of vomer.
19. Septum.
20. Presphenoid.
21. Respiratory tract in the posterior inferior part of the nares.

PLATE X.

Transverse sections through both nasal fossae of the dog, cut slightly obliquely so as to pass through different parts of corresponding structures in each fossa, looking anteriorly. Text as in Plate IX.

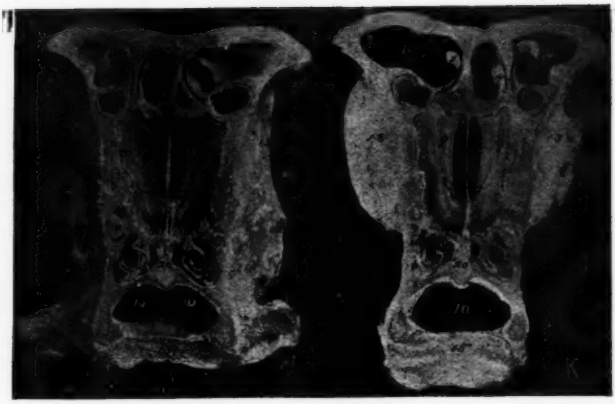

PLATE XI.

Transverse sections through both nasal fossae of the dog, cu slightly obliquely so as to pass through different parts of corresponding structures in each fossa. J. looking anteriorly, K. posteriorly. Text as in Plate IX.

PLATE XII.

1. Frontal bone, cancellous.
2. Cribriform plate.
3. Anterior etmoidal cell.
3.' Posterior ethmoidal cell.
4. Ethmoidal turbinal.
5. Ethmoidal turbinal extending into sphenoidal sinus.
6. Sphenoidal sinus.
7. Maxillary turbinal.
8. Brain cavity.

The turbinals are comparatively simple structures. The maxillary sinus is large and its wall are smooth, except the external lateral wall which presents a decided ridge extending upward to the ethmoidal bone.

The frontal bone is cancellous and there is no true frontal sinus but the anterior ethmoidal cell (3) extends up into the frontal bone.

The posterior end of the ethmoidal turbinal (5) extends into the sphenoidal sinus and divides it into two distinct cavities. The smaller, anterior cavity communicates with the superior meatus through the posterior ethmoidal cell (3'). The larger cavity (6) occupies the greater part of the sphenoidal bone and opens into the superior meatus under that part of the ethmoidal turbinal which extends into the sphenoidal sinus.

PLATE XIII.

1. Frontal sinus.
2. Cribriform plate.
3, and 4. Ethmoidal turbinal. The ostium of the middle ethmoidal cell is just below the figure 3, and the ostia of the sphenoidal and posterior ethmoidal cells are below the figure 4.
5. Superior sphenoidal sinus.
6. Inferior sphenoidal sinus.
7. Bony ridge on external lateral wall of maxillary sinus.
8. Brain cavity.

In this specimen the maxillary and the inferior part of the ethmoidal turbinal have been lost.

The frontal sinus is large and extends over the orbit; it drains into the anterior ethmoidal cell, which opens into the middle meatus.

The maxillary sinus is large and is partially sub-divided by a bony ridge extending upward and backward across its external latteral wall and roof, and uniting with the ethmoidal bone. This ridge increases in thickness as it ascends and gives off several lateral branches and corresponds to the ethmoidal turbinal which extends down into the maxillary sinus in the dog.

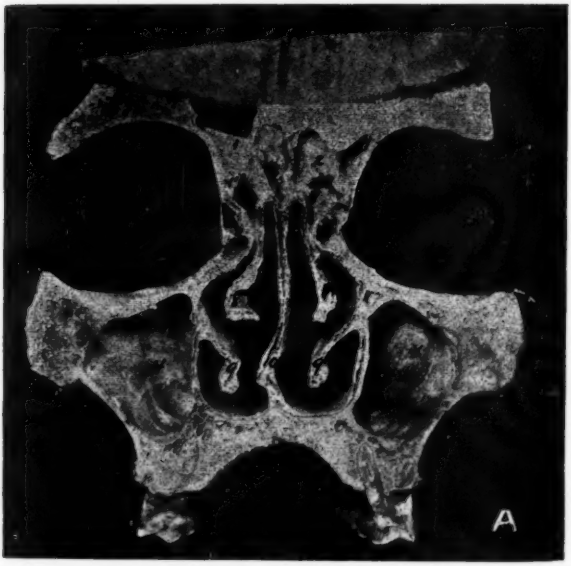

PLATE XIV.

Section through an adult human skull, looking anteriorly.

Text for this plate and the three following:

1. Frontal sinus, opening into a small ethmoidal cell 1', and thence into the middle meatus.
2. Large anterior ethmoidal cells extending upward into the frontal bone.
3. Ethmoidal cells.
4. Maxillary sinuses.
5. Bony partition in maxillary sinus.
6. Large ethmoidal cells extending into sphenoidal bone.
7. Sphenoidal sinuses. The ostiasphenoidalis are just below the figures.
8. Ethmoidal turbinals.
9. Maxillary turbinals.
10. Septum.

This skull shows several interesting rudimentary structures. The left frontal sinus (Plate XIV and XV, 1) communicates with the nasal fossa through an ethmoidal cell. There is no frontal sinus in the right half of the skull but the anterior ethmoidal cell on this side extends up into the frontal bone (Plate XIV and XV 2).

The left maxillary sinus is partially subdivided by a bony partition (Plate XV. 5) which corresponds to the partition usually present in the maxillary sinus of the monkey. One of the large ethmoidal cells contains a distinct turbinal scroll; unfortunately this scroll is in such a position that it does not appear in any of the photographs.

The left sphenoidal sinus is divided into a superior and inferior cavity by a thin bony partition (Plate XVII 6 and 7). This partition is distinctly an ethmoidal structure and springs from the superior ethmoidal turbinal. Therefore I have designated the superior cavity as an ethmoidal cell and also because its anterior part is entirely surrounded by the ethmoidal bone and its ostium is on the external side of the superior ethmoidal turbinal in common with other ethmoidal cells. This bony partition in the sphenoidal sinus corresponds to the extension of the ethmoidal turbinal into the sphenoidal sinus in the orangutan) Plate XII 5).

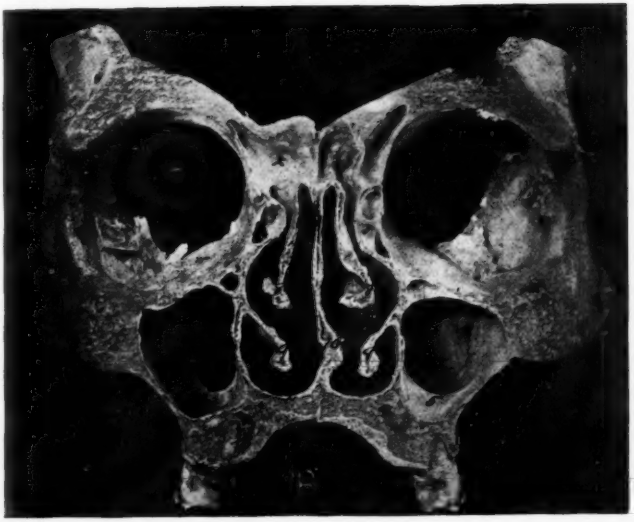

PLATE XV.

Section through the same skull, looking posteriorly. Text as in Plate XIV.

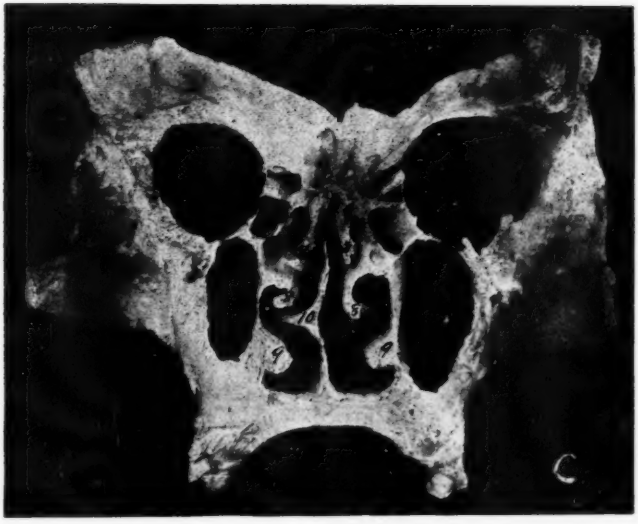

PLATE XVI.

Section through the same skull looking anteriorly. Text as in Plate XIV.

PLATE XVII.

Section through the same skull looking anteriorly. Text as in Plate XIV.

XXIV.

FINAL HISTORY OF A SUPPOSED CASE OF "VOCAL NODULE"—EPITHELIOMA OF THE LARYNX—THYROTOMY—DEATH.*

By Chas. H. Knight, M.D.,

NEW YORK.

At a meeting of this association in 1901 I read a paper entitled "Vocal Nodules," suggested by a supposed case of the disease, the history of which was given in detail. The outcome of the case was so unexpected, and in many respects so instructive, that it seems desirable to place on record its subsequent history.

At the time of my first report laryngeal symptoms had been present more than a year, although the "nodule" had been discovered only about nine months previous. During the summer of 1901, the following winter and the summer of 1902 every effort was made to give the patient the benefit of the best possible surroundings, and the local treatment was restricted to the use of sedative and mild astringent sprays. The voice continued hoarse, with occasional intervals of partial recovery, until aphonia became practically permanent. In October, 1902, the "nodule" had increased slightly in size and prominence, but there was no obvious infiltration of the vocal band or loss of power. There was no pain and there were no glandular enlargements. The patient was extremely discouraged, although her general

*Read at a meeting of the American Laryngological Association, June 4, 1904.

condition had decidedly improved. Incidentally it may be mentioned that from the first she was possessed with the notion that she had cancer and could not be cured. She would be reassured, but now and again reverted to the idea.

About this time she saw several of my colleagues, one of whom removed a fragment of the projecting mass with a cold wire snare. The microscopic examination of this fragment (about one-sixteenth of an inch in thickness) made by an experienced pathologist, showed that the tissue was composed almost solely of epithelial cells having the appearance of a papilloma. The arrangement of the cells gave no evidence of rapid growth. So little submucous tissue was present that no accurate idea of the depth of the cell layer could be formed, but no indication of invasion of the submucosa were seen. The microscopic diagnosis was "papilloma of larynx, malignant tendencies." The latter was based on the following three features; (1.) Unusual amount of chromatin in the nuclei. (2.) Concentric arrangement of cells in whirls or groups of 10 or 12. (3.) Some cell inclusions resembling those seen in carcinoma. There followed an excessive degree of reaction which did not subside. The vocal band became swollen and red, and the neoplasm showed a tendency to creep toward the anterior commissure. Accordingly it was decided to do a laryngo-fissure and let further interference be subject to the state of things thus disclosed. On December 7th, 1902, a thyrotomy was done in the most skillful manner by one of our leading surgeons. Pieces of affected tissue were removed and at once submitted to an expert microscopist, who, after having examined several sections pronounced the lesion an epithelioma, "all the sections showing extensive infiltration." On the strength of this finding all the soft tissues, including the arytenoid of the right side of the larynx were resected. The cavity was left open, packed lightly with gauze, a long tube having been left in the trachea, and the patient was removed from the table in a most excellent condition. She was left in charge of two experienced nurses, with every appliance at hand to meet emergencies, and one of the house staff remained with her until after complete recovery from ether. The operation itself was free from complications and was rapidly done,

having been interrupted for about fifteen minutes while the microscopic examination was in progress. The patient passed a quiet afternoon, except for some restlessness at first, and finally fell asleep. Toward evening she suddenly awakened, sprang up in bed, gasping for breath, became cyanosed and presently fell back unconscious. The nurse called a surgeon who reached the bedside almost immediately, at once extracted the tracheal tube, which was apparently in proper place, catheterized the trachea, did artificial respiration and resorted to every possible means of resusitation without avail.

The operating surgeon has courteously placed at my disposal the following comments: "Whether her death was due to a slipping of the tube from the trachea or to pulmonary embolism I am unable to say. I am inclined to believe the former, al hough both nurses state that up to the moment of her apparent suffocation she had been sleeping quietly, had not coughed, and had made no unusual effort which would have been likely to displace the tube. Moreover, shortly after her return to her room she had vomited slightly and had coughed at intervals without producing such a result."

This case enforces certain lessons that have been often taught, and unfortunately it brings us no nearer the solutiyn of several questions many times asked:

(1.) The stimulation to activity of malignant disease by endolaryngeal manipulations has been repeatedly noted. Here it was repeatedly marked. We should always be prepared for this event and if possible have the full consent of the patient to resort to whatever further operative procedures may seem advisable.

(2.) The testimony of the microscope is not to be depended upon unless the whole thickness of suspected tissue at several points can be secured. Numerous cases are on record in which the microscope has antagonised the clinical history, and others in which a microscopic diagnosis has not been verified. It is seldom possible to obtain a satisfactory microscopic specimen by means of forceps passed through the mouth.

(3.) The question of transformation of a benign growth into one of malignant character has apparently been decided

by statistics in the negative, yet the fact remains that in this instance the appearance and history of the lesion continued absolutely benign for more than two years. In many cases a much longer period of quiescence has been observed. There was nothing whatever in the course of the disease to justify a suspicion of malignancy until after the use of the snare. It is not supposed that the latter did more than excite the virulence of the morbid process.

(4.) We are still in the dark as to positive signs by which to identify malignancy at its inception. Was this growth benign at the outset, and if so when did the malignant change occur?

At present the only way in some cases in which to determine the character of a doubtful laryngeal lesion is by its complete exposure through an external wound, and even then, in the words of Butlin: "It may be difficult to say what the nature of the disease is."

The picture in the laryngeal mirror is often indecisive as to the character of a lesion and mi leading as to its extent. Almost always in splitting the larynx we find much more territory involved than was previously suspected. The prognosis in *intrinsic* cancer of the larynx is perhaps more favorable than that of malignant disease in almost any other region of the body, yet our guiding principle here as elsewhere should be early operation to include by a wide margin every vestige of morbid tissue.

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XXV.

MALIGNANT GROWTHS AT THE BASE OF THE TONGUE.

By Richard H. Johnston, M.D.

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To those of us interested in pathology it has no doubt seemed strange that "Waldeyer's Ring," should be singularly free from the formation of malignant growths. Especially is this true of sarcoma. In medical literature very few cases of this form of tumor affecting the tonsillar tissue have been reported. I have never seen a case clinically, but have had an opportunity of making a histologic examination of a sarcoma of the lingual tonsil which was referred to me by Dr. John R. Winslow in whose practice the case occurred. The notes of the case were also furnished me by him. The patient was a male of advanced years who had a large swelling at the base of the tongue. The tumor was hard and non-movable. There was no ulceration. Removal was successful with the cold snare. Hemorrhage was slight. The tissue removed was somewhat larger than a pigeon egg. Evidently the greater part if not all of the tumor was removed. After hardening in alcohol sections were cut and stained in hematoxylon and eosin. The histologic formation of the growth follows.

Under the low power the tumor is seen to consist almost

entirely of cells. In certain areas connective tissue bands are evident. There is a decided infiltration of the surrounding normal tissues with cells but the neoplasm is fairly well circumscribed.

With the high power the cellular mass is much more prominent. Some of the cells are as small as small lymphocytes while others are large in size. The shape of the cells varies slightly from the round to the ovoid. No spindle cells are detected in the tumor. Some of the cells are multinuclear, while most of them possess a single, distinct nucleus. The protoplasm does not stain as deeply with the hematoxylon as the nuclei. The connective tissue is present only in certain areas. It consists of wavy fibres rather thin. Blood vessels are rather numerous. Some are large while the majority are small in calibre. The smaller vessels are probably newly formed in the tumor mass. The walls of the blood vessels are normal in thickness and are lined with flat endothelium. The tumor may be described as a round cell sarcoma consisting of small and large cells.

The growths most likely to be confounded with sarcoma in this location are tuberculoma and syphiloma. If the tumor is non-ulcerating, epithelioma can almost certainly be eliminated. The presence of giant and epithelioid cells along with tubercles and possibly tubercle bacilli are characteristic of tuberculosis. In gumma, the diagnosis can nearly always be made from the changes in the vessel walls, especially the arteries. These show a decided thickening from endarteritis. In the section above described there were no changes in the vessel walls.

The second tumor was observed in a man of 57 years. Six months before he came to the hospital for treatment he was annoyed by the sensation of a foreign body in his throat whenever he swallowed. He consulted a physician at once and was told by him after an examination of the throat that he had a small tumor at the base of the tongue. He was given potassium iodid in increasing doses, but the swelling continued to grow. At the time of his entrance into the hospital he was suffering great pain which radiated into both ears. There was no difficulty in talking, but the throat was filled with a thick, tenacious mucus which was

very hard to expel. The anterior, cervical glands were enlarged but not painful. No history of syphilis could be obtained. The examination of the throat showed a large swelling at the base of the tongue, which obliterated all traces of the lingual tonsil. The outer surface of this tumor was deeply ulcerated, which gave the tongue the appearance of a cauliflower. The ulcer was covered by a grayish exudate, which, when wiped away, revealed a sloughing base. The odor from the growth was very offensive.

Feeling satisfied from the history of the case and the appearance of the growth that it was malignant and probably epitheliomatous, I removed a small piece for microscopic examination. The patient was given palliative treatment until the diagnosis could be made with certainty. The examination of the section the following day resulted in the positive diagnosis of epithelioma. The outer surface of the section was covered with squamous epithelium. Extending down from this were processes of irregularly shaped epithelial cells. These processes were of different shapes and different lengths, some large others small. At certain points there was entire cutting off of the epithelial processes from the main mass, giving rise to islands of cells at these points. In some of these collections of cells were to be seen the so-called nests which are characteristic of this form of tumor. These nests in a modified degree are sometimes seen in normal epithelium or in benign papillomata, but they differ very materially from the true epitheliomatous nests and to the experienced eye there is no difficulty in distinguishing between the two conditions. The entire section contained these collections of epithelial cells.

The clinical diagnosis was confirmed by the microscope. Since at the time o the examination there was no involvement of the epiglottis or of the walls of the pharynx, the tumor must be considered a primary epithelioma of the base of the tongue or of the lingual tonsil. No operation was advised on account of the extreme weakness of the patient and because of the extent of the glandular involvement. At a later stage of the disease the epiglottis and the walls of the pharynx became involved and the patient died three months later, just nine months from the beginning of his symptoms.

This case is offered for publication for two reasons. In the first place primary epithelioma of the base of the tongue is very rare. Butlin, in the collection of 80 cases of carcinoma of the tongue, found only one at the base. In the second place it emphasizes the great importance of an early diagnosis in such cases. This can be done only with the help of a careful microscopic examination. In this case, if the histologic examination had been resorted to instead of saturating the patient with potassium iodid when it was evident that the drug was doing no good, it is highly probable that the disease could have been completely eradicated or the patient's life prolonged. Had the patient fallen into the hands of a physician who appreciates the value of the microscope and its limitations, the result would have been different. I have no hesitation in saying that the microscope is a powerful means of diagnosis in these doubtful cases and should be resorted to in the beginning of the disease. Any physician who does not observe this rule has not done his full duty to his patient.

The extreme rarity of sarcoma of the tongue justifies the report of the first case. In the literature I have found only 25 cases recorded and some of these were probably not sarcomata. Five of these are described as having been located at the base of the tongue. All of these were of the round cell form or lymphosarcomata. The prognosis is not so unfavorable if the growth can be removed in the beginning. In the five cases recorded in the literature, to which must be added a sixth by Prota, there were four recoveries following operation and two deaths. Prota's case was a fibrosarcoma. The ages of the patients ranged from 19 to 56 years.

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XXVI

CONCERNING THE SINUS FRONTALES IN MAN WITH OBSERVATIONS UPON THEM IN SOME OTHER MAMMALIAN SKULLS.*

By Adelbert Watts Lee, M. D.

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A review of the literature suggests the necessity of a more complete description of the sinus frontales with some revision of the nomenclature according to their developmental characteristics, as well as a complete study of their architecture. The writer accordingly took Dr. Flint's suggestion to make a series of metallic injections of these cavities in order to reveal in positive form the normal negative outlines of the sinuses. Since students rarely obtain a stereoscopic concept of the pneumatic spaces contributing so markedly to the shape of the human cranium, an employment of the method by classes engaged in routine dissections is urged. In this laboratory it is found that students gain a much clearer idea of the cavities when they are studied by means of lead casts.

As will be seen by reference to the illustrations, two kinds of technique were utilized in gathering data for this paper, namely, chiselling and casting. The chisel calls for time and some delicacy of manipulation to get good results, but casting is simple, rapid, and seems by far the best method, inasmuch as the lead mold positively expresses the negative sinus. Following is an outline of the casting method:

When students have completed the dissections of the external face and cranium, a sagittal section of the head is made corresponding to the median line. While one or the other sinus frequently overlaps the median plane, the septum

*From the Hearst Anatomical Laboratory of the University of California.

*Reprinted by courtesy of Johns Hospital Bulletin.

between them is commonly complete; accordingly it occasionally happens that the sections open only one sinus, and a pour-hole into the other must be provided by drilling through the intact septum. To prevent the metal escaping from the sinus by way of the infundibulum ethmoidale, the hiatus semilunaris is exposed and plugged with moist clay. In a melting pot over a circular gas burner, lead is raised to a temperature of brisk oxidation, as metal of a lower temperature often so idifies before reaching the utmost limits of the cavity. With these precautions the metal is directed into the sinus with a pouring ladle. It must, however, be poured slowly and in a thin stream, for should it enter rapidly and in a large mass, air is often retained in the sinus in quantities sufficient to ruin the cast. After allowing the lead to cool thoroughly the material is returned to the student for further dissection. Under the supervision of an instructor, he carefully removes the molds. They are then marked with the body number, making it possible at any time to obtain collateral information concerning the individual by consulting the records in the preparation room. The casts have rough surfaces due to generation of gas by the melted lead coming in contact with organic tissue in the cavities. Furthermore, the metal contracts slightly while cooling, but in the main the molds approximate quite exactly the sinuses in which they are made.

This study is based upon examination of twenty-one adult male subjects, in age ranging from twenty-five to sixty years. The molds of all the sinuses changed from their normal state by pathological processes were rejected.

The cavities are said to be generally smaller in the female than in the male. Owen* has observed that the sinuses of Europeans are larger than in some other tribes. He states that they are imperfectly developed among the native Australians, and in consequence this deficiency engenders a want of resonance to the voice. In opposition to this, Macalister

*Owen Ours: Circle of Sciences, p. 167. Quoted by Humphry. A Treatise on the Human Skeleton, etc. G. M. Humphry. Macmillan & Co., London, 1858.

states that the sinuses are very large in Australian crania,* and Hilton finds them quite extensive in the skull of an African aborigine.† The latter, however, made but a single observation, and it is possible, of course, that his specimen might have been an unusual one.

Sufficient attention has not been called either to the importance of symmetry in the architecture of living forms, to its significance, or to the means by which it becomes one of the most striking characteristics of all the higher forms of organic life. It is suggestive, however, that all structures in the body are at some period in the life-history of the organism either completely or partially symmetrical—that is to say, form either complete mirror changes of each other, or may be divided by a median plane into halves which are enanteomorphic. In this connection it is noteworthy, therefore, that the sinus frontales vary in respect to symmetry more than any other paired structures in the human body. This is noted not only with reference to the deflection of the septum sinuum frontalium but also in other characteristics of sinuses in the same skull.

The sinuses from two cavities in the os frontale are situated dorsad of the areus superciliaris and cephalad of the orbita, displacing the spongy bone between the inner and outer tables. They communicate with the meatus nasi medius by means of an osseous tube, the infundibulum ethmoidale, and are lined with a continuation of the nasal mucous membrane. Their position divides them into two parts, the pars frontalis, the portion of the sinus in the squama, and the pars orbitalis, the portion in the pars orbitalis ossis frontalis. Both frontal parts are shown in Fig. 5 *P. F. D.* and *P. F. S.* The orbital parts are shown in the same figure, *P. O. D* and *P. O. S.* Between the partes frontales is a perforate or imperforate partition of bone. leaning to one side or the other, the septum sinuum frontalium (Fig. 5, *s. s. f.*). The

*A Text Book of Human Anatomy, etc. A Macalister. P. Blakiston Son & Co.

†Hilton On the Cranium, p. 13. Quoted by Humphrey. A Treatise on the Human Skeleton, etc. G. M. Humphrey. Macmillan & Co., London, 1858.

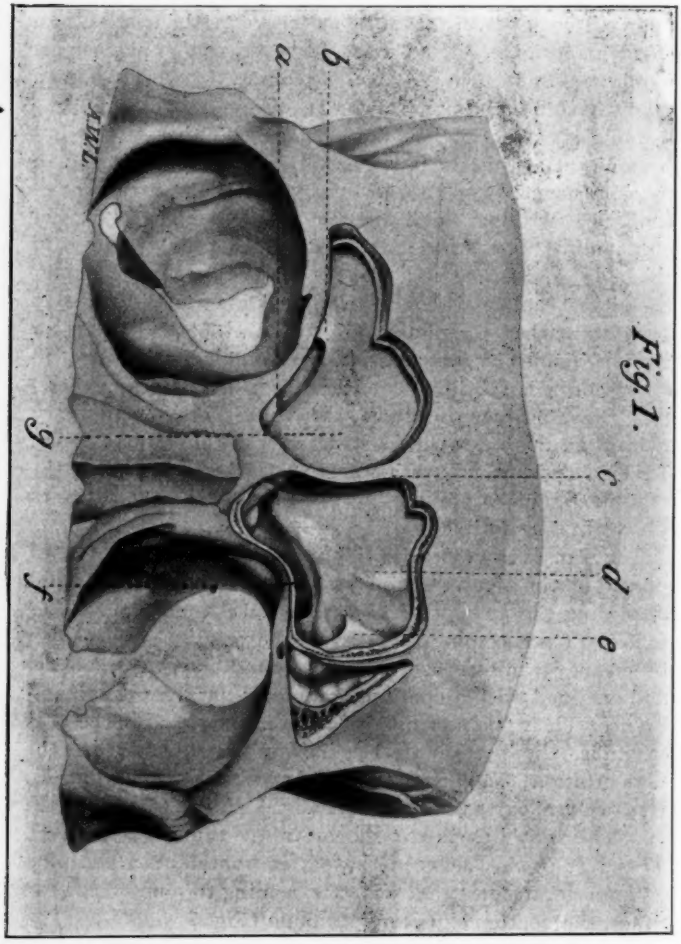

Fig. 1.

Figure 1.—Human Sinus Frontalis. In this case the arcus superciliaris on each side was equally developed, notwithstanding the difference in size of the sinuses. The median septum approximates the mid-line more clearly than is usually the case.

a—Summit of Bulla ethmoidalis dextra. Its fellow is opposite

occupying a slightly lower level.

b—Septum interpartes.

c—A strip of the outer table of the skull and a portion of diploe above the septum sinuum frontalium. The latter is intact and very thin. This septum more nearly approximates the median line than is usually the case.

d—Linear elevation of the dorsal wall indicating a poorly developed septum lateralis.

e—Septum lateralis.

f—Pars orbitalis sin.

g—Pars orbitalis dex.

frontal and orbital parts of the cavity are separated from each other in varying degrees by a partition of bone called the septum interpartes (5, *s. i. d.*). The cavities are also subdivided by bony trabeculae called septa laterales when in the pars frontalis (Fig. 5, *s. l. s.*), and septa dorsales when in the pars orbitalis (Fig. 5, *s. d. s.*). The lateral limits of the sinuses are ordinarily distant from each other about seven centimeters. They reach cephalad into the squama about three centimeters, and the diameter of a sinus from the most ventral point of the pars frontalis to the most dorsal one of the pars orbitalis is about two and a half centimeters. These figures of course represent the average of the twenty-one specimens used for this study. The pars frontalis of one sinus usually overlaps the median line and encroaches upon the territory of the other. This unequal development is responsible for the deflection of the septum sinuum frontalium. The two cavities always show a marked difference in outline and capacity, as is readily seen upon inspection of the casts represented in Fig. 5.

The sinus frontalis may be absent throughout life. This condition was found once in the skull of an old adult and the usual superciliary eminences were entirely wanting on both sides.* The extremes in size are pictured in Figs. 3 and 4, and 4, the former showing the largest, the latter the smallest casts in the series.

Contrary to the statement of Sappey,† examination of the

*A series of sagittal sections found this os frontale of an unusual construction; it was thin throughout; the diploe included between the outer and inner tables was reduced to a thin line.

†Traite d'Anatomie Descriptive, Ph. C. Sappey, Vol. 1, p. 264. Paris, 1888.

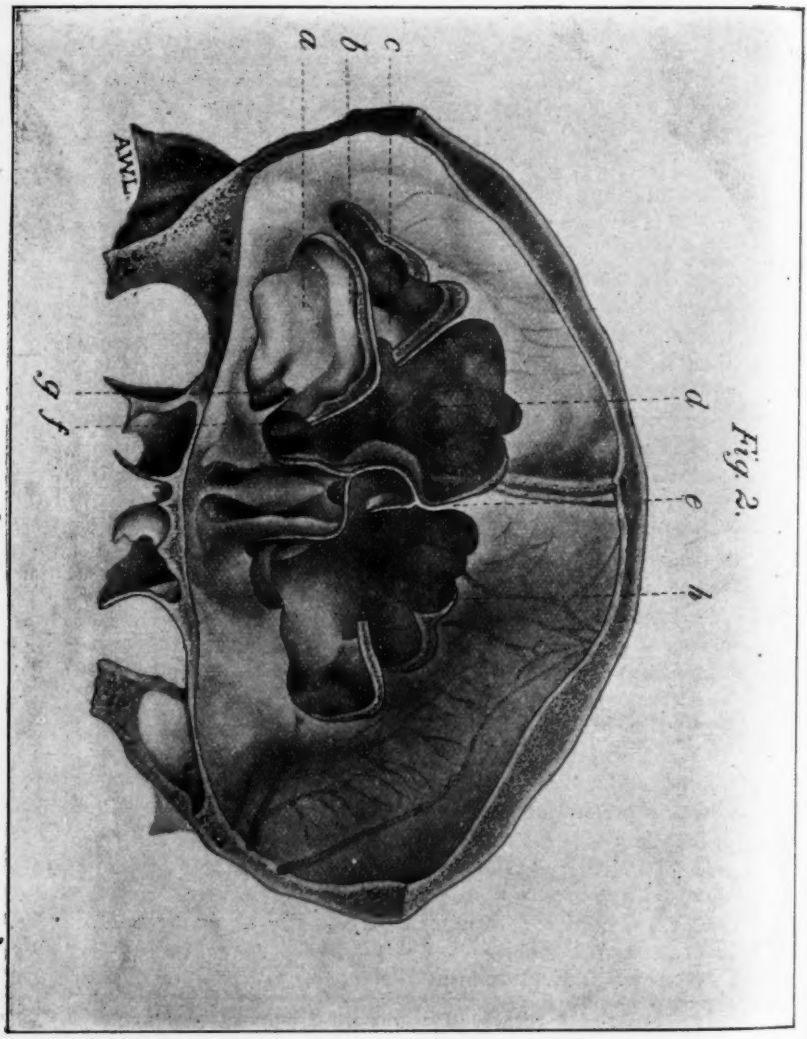

Fig. 2.

Figure 2.—Nearly all of the bone dorsad of the sinuses is removed. The left sinus is considerably the largest.

a—Pars orbitalis sin.

b—Long, tortuous and thin septum interpartes sin. Its fellow is more ventrally placed, is shorter, thicker and stands in one plane.

c—Septum lateralis sin.

d—Pars frontalis sin.

e—A strip of the inner table of the skull and a trace of diploe corresponding to the septum sinuum frontalium, although the latter is very thin. In this specimen the septum is intact and is in its characteristic, deflected position, but the median chamber of the right sinus has caused an unusual division of the septum, one arm being continuous with the medial walls of the cavities and the other arm ending by a free, caudal border in the pars frontalis dex.

f—Bulla ethmoidalis sin.

g—Septum dorsalis sin. It is longer and thinner than usual. Opposite is seen another septum dorsalis stretching between the dorsal and medial walls of the pars orbitalis dex. and has a free, falciform, cephalic border.

h—Linear elevation of ventral wall and is all that remains of a one-time septum lateralis.

arcus superciliaris develo ps no precise information concerning the size of the sinus beyond. The arcus may be larger or smaller than the sinus. In the majority of cases, it is quite true that a large superciliary prominence indicates a large cavity behind it and also the reverse, but the presence of an arcus is no guarantee of a sinus in the squama ossis frontalis. One old individual of the series possessed strongly fashioned eminences above the orbits, but had sinuses extending only into the orbital roofs. Casts of these appear in Fig. 4.

Between the sinuses is the septum sinuum frontalium. Proportionate to their extent into the squama. the septum is contracted in breadth and increased in length. It seldom approximates the median line, a fact due to the unequal size of the cavities. When the two sinuses communicate the septum may be perforated at any point in its extent. Usually, however, if not intact, the opening cuts through its caudal extremity. Out of twenty pairs the right sinus was largest in thirteen, the left one in three, and the remaining four were about equal.

Since in the process of their evolution the sinus frontales extend into both the squama and pars orbitalis ossis frontalis, it may be convenient to classify them according to their position; any pair may consist of partes orbitales, or partes

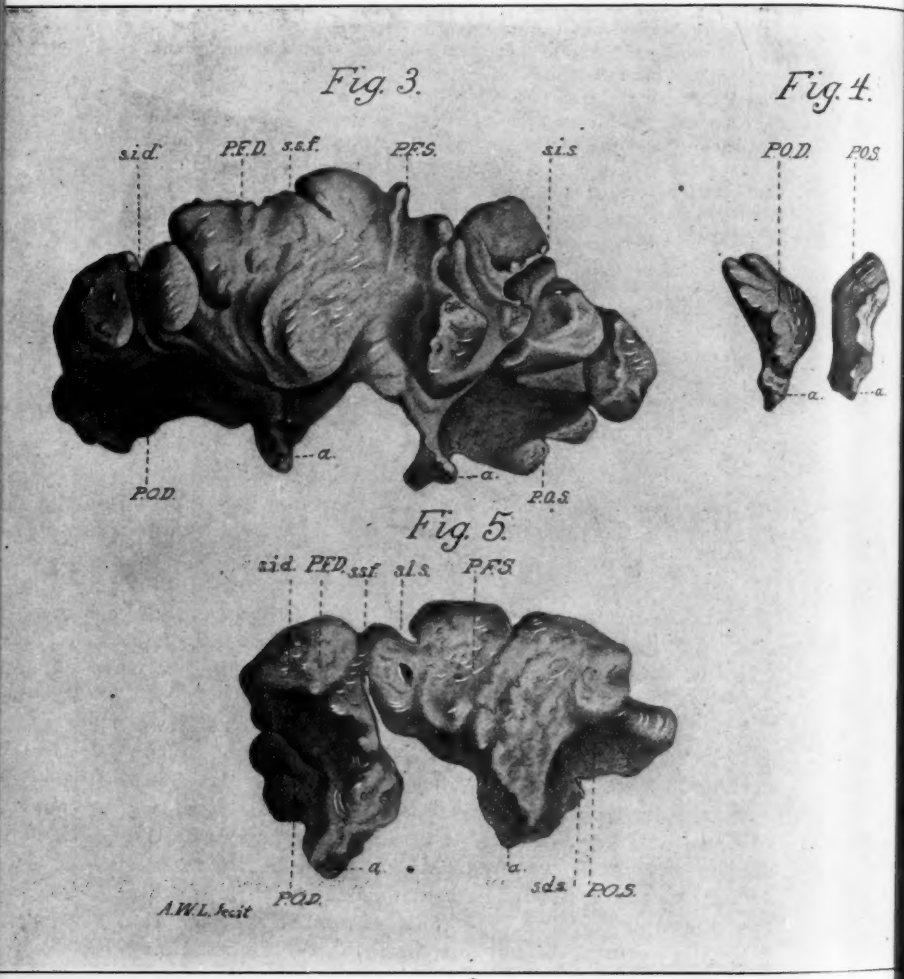

Figure 3.—A Wood's metal cast of a large sinus (natural size).
P. O. D.—Pars Orbitalis Dex.
P. F. D.—Pars Frontalis Dex.
P. O. S.—Pars Orbitalis Sin.

P. F. S.—Pars Frontalis sin.
s. i. d.—Septum interpartes dex.
s. s. f.—Remains of septum sinuum frontalium.
s. i. s.—Septum interpartes sin.
a.—Metal prolongation into the cephalic end of the infundibulum ethmoidale.

Figure 4.—Lead casts of a small sinus and representing an instance where the sinuses were purely orbital in development (natural size).

P. O. D.—Pars Orbitalis Dex.
P. O. S.—Pars Orbitalis Sin.
a.—Metal prolongation into the cephalic end of the infundibulum ethmoidale.

frontale, but the latter condition is much more common. If the sinus of one side presents both parts well developed, its mate is usually approximately the same in form. However, when one sinus shows a capacious pars frontalis with only a slight orbital extension, the opposite cavity as a rule will be purely frontal in location.

The pars frontalis is, at times, no more than a slight, cephalic dilation of the infundibulum ethmoidale, reaching not further than the caudal level of the glabella (Fig. 8, *A*). While again it will be seen passing cephalad to the caudal circumference of the tuber frontale, stretching laterad to the linea temporalis, or even, according to Hajek,* excavating the processus zygomaticus and running dorsad to the sutura sphenofrontalis.

Out of nineteen cases where it appeared on both sides the pars frontalis was largest in thirteen. This frontal portion of the sinus (Fig. 1, *g*), whether large or small, is commonly a single chamber, but it consists frequently of a series of unequally-sized pockets, partially separated from each other by bony septa. These septa laterales (Fig. 1, *e*) are hung from the cephalic junction of the ventral and dorsal walls of the sinus and, before extending far into the sinus, often end in free, falciform borders. Occasionally they are so long that the several chambers are continuous with each other by very small openings. The septa, however, do not always span the intermural space but exist as low lineal elevations,

*Nebenhölen der Nase. Hajek. Leipzig und Wien.

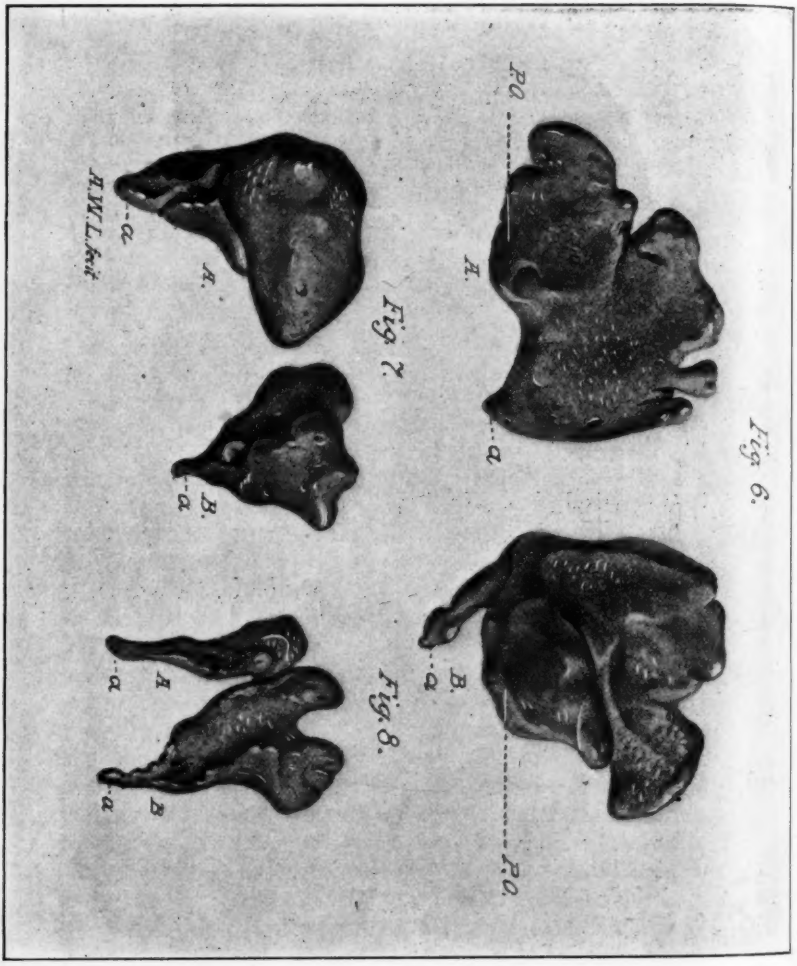

Figure 5.—Casts of an average pair of sinuses, showing a natural perforation of the septum sinuum frontalium (natural size).

P. O. D.—Pars Orbitalis Dex.

P. O. S.—Pars Orbitalis Sin.

P. F. S.—Pars Frontalis Sin.

s. i. d.—Septum interpartes dex.

s. s. f.—Septum sinuum frontalium.
s. l. s.—Septum lateralis sin.
s. d. s.—Septum dorsalis sin.
a. Metal prolongation into the cephalic end of the infundibulum ethmoidale.

Figure 6.—A and B are casts from different subjects (natural size), dorsal view. They illustrate the variation in position of the partes orbitales. P. O. of A. shows a left orbital extension proceeding from the lateral portion of the pars frontalis, while P. O. of B. is a pars orbitalis dex., resembling an outstretched wing, passing from the medial portion of the pars frontalis. A long septum interpartes partially separated the masses of the metal representing the two divisions of the sinus.

Figure 7.—A and B are casts from the left sinuses of different subjects (natural size), ventral view. A shows no lateral septum, while B, although a smaller sinus, is nearly bisected with an interval created by a long septum lateralis, indicating that these structures have no direct bearing on the size of the sinus.

Figure 8.—A and B are casts from a middle-aged adult (natural size), ventral view, showing one sinus as a simple dilation of the cephalic end of the infundibulum ethmoidale. The casts A and B are joined by a mass of metal filling an opening in the middle third of the septum sinuum frontalium.

in the majority of instances on the vent al wall (Fig. 2, *h*, and Fig. 1, *d*). It is the rule for the septa laterales to increase in number with an increase in the lateral extent of the sinus, but the amplitude of the latter, however, bears no definite relation to the presence of the septa. A large cavity may exist without them (Fig. 7, *A*), while a comparatively small one may possess a number. The capacity of a sinus, moreover, does not seem to influence the form of the septa. In a large cavity the one existing partition is often found so thin that light passes through it almost unobstructed, while those of a smaller sinus may be short and thick.

The type of sinus represented by the pars orbitalis alone often exists on one or both sides. This, however, is much less common than the purely frontal type, or a combination of the two. The pars orbitalis is represented at times by a simple, slight, dorsal extension of the pars frontalis, while at others it reaches out over almost the entire orbital roof. In the series, the pars orbitalis existed bilaterally in ten crania, where that of one side showed no marked size difference with its fellow. Out of twenty-six single sinuses, however, pre-

senting a combination of orbital and frontal parts, the latter was most voluminous in twenty-two.

The pars orbitalis (Fig, 2, *d*) usually passes dorsad from the medial portion of the pars frontalis (Fig. 6, *B. P. O.*), nevertheless it might be an extension from the lateral part of the frontal division of the sinus (Fig. 6, *A. P. O.*). At times both parts communicate with each other very freely, but they usually are divided by a septum reaching medially

Fig. 9.

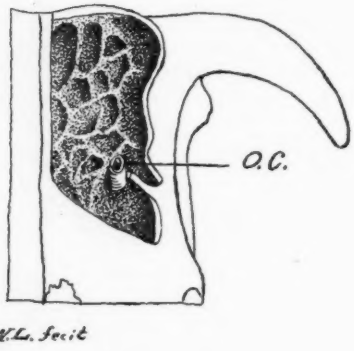

Figure 9.—Detail of an illustration from Ellenberg and Baum, showing the immense sinus frontalis of the ox, which excavates the frontal, parietal and occipital bones, as well as the horn.

from the lateral limit of the sinus. This septum interpartes (Fig. 2, *b*) may exist in one sinus and be absent on the opposite side, or as is usually the case, be present in both. In construction it often varies from a long, thin partition to a short, thick one. Like the frontal division, the pars orbitalis may consist of one or many chambers, but its pockets are seldom as large, since the septa forming them are, as a rule, rarely so deep as the septa laterales. The septa dorsales, subdividing the orbital portions of the sinus, are usually,

however, short and thick. Generally speaking, they arise from the lateral and dorsal limits of the pars orbitalis, although they may sometimes pass laterally from the median wall.

The sinus frontalis of the ox excavates the frontal, parietal and occipital bones, making a very large chamber between the inner and outer tables which may or may not communicate with its neighbor on the opposite side. The two tables of the cranium are not reinforced by complete bridges spanning the sinus, except around the peripheral portions of the cavity, where thick trabeculae divide it into small pockets, not comparable in size, however, to similar structures in the human skull. The sinus extends up into the horn which near its base is also trabeculated. Dorsad of the horn there is a system of trabeculae admirably situated for the reinforcement of this part of the skull against strains or shocks imparted through the horns. These also form with the outer table of the skull an eminence for the attachment of the large cervical muscles. That the presence of the sinus in this animal affords some protection against traumatism, is shown by a specimen with an extensively healed fracture of the outer table over the sinus, while the inner table remains intact. It does not seem possible to subdivide the sinus frontalis in the ox's skull into distinct portions, unless they are named arbitrarily from the bones or regions in which they occur.

The frontal sinus in the pig's skull (Fig. 10) excavates the frontal and a portion of the parietal bone. It does not however, extend into the occipital bone as is the case with the ox. This sinus is usually separated from its neighbor by a complete bony septum somewhat more regularly placed than the septum sinuum frontalium in the human cranium, although the partition in the pig may have usually a considerable lateral deflection. This sinus is often subdivided into a pars medialis and a pars lateralis by a septum interpartes which extends anteriorly to a greater or less extent. These two parts, however. do not correspond respectively to the pars frontalis and pars orbitalis of the sinus frontalis in the human cranium. The par medialis is included between the median and lateral partition and corresponds to the pars

frontalis, while the pars lateralis is located between the orbit and the septum interpartes corresponding to the pars orbitalis of the human sinus frontalis. The two portions of the

Fig. 10.

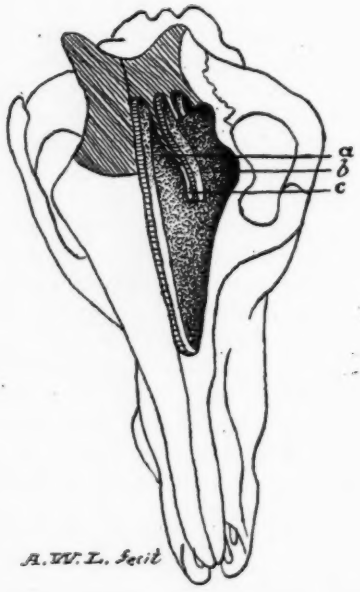

Figure 10.—A preparation of adult pig's skull viewed slightly perspective. The area shaded with diagonal lines represents a portion of bone removed to exhibit the sinus which excavates a position in the frontal and parietal bones. Here the cavity is differentiated into a pars medialis (*a*) and a pars lateralis (*b*) by a long septum interpartes (*c*).

sinus communicate anteriorly. As in the human and other specimens, the sinus is subdivided around its periphery by a series of small trabeculae into small pockets. These, however, cannot lend a great deal of additional strength in re-

inforcing the vault of the cranium, but the septum interpartes (Fig. 10, *c*) reaching from the outer to the inner table and extending forward through the broader part of the sinus contributes a great deal to its structural strength. The sinus

Fig. 11.

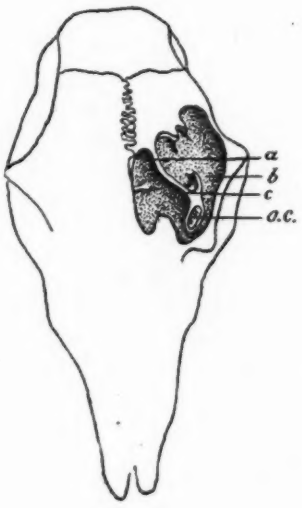

Figure 11.—A preparation of an adult sheep's skull, where the sinus frontalis is confined to the frontal bone.
a—Pars medialis.
b—Pars lateralis.
c—Septum interpartes.
The latter in this animal has the additional function of lending support to the osseous tube O. C., which transmits the superciliary vessels and nerve.

frontalis in the sheep is limited to the ventral and lateral portions of the ossis frontalis. Its depth in the maximum is from 0.7 to 1.5 centimeters. Like the sinus in the pig's skull, it is subdivided into two portions by a septum interpartes

and the trabeculae around the periphery of the sinus are much more developed, exaggerating the sacculation of the sinus to even a greater degree than is shown in Fig 11.

In the dog the frontal sinus (Fig. 12) shows an irregularly spherical or ovoid cavity in the frontal bone, usually com-

Fig. 12.

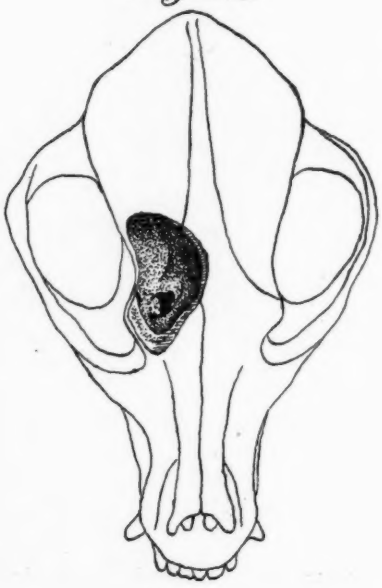

Figure 12.—A preparation of an adult male dog's skull, and shows the sinus entirely within the frontal bone.

pletely separated from its mate by the septum. No trabeculae bridging the tables are found in this skull and those around the periphery of the sinus are only developed to the extent of slight ridges, thus causing a slight sacculation of the periphery of the sinus. In the skull of the cat (Fig. 13)

the sinuses are limited to the frontal bone and are completely separated from each other by the septum sinuum frontalium. This cavity can often be divided into the pars frontalis and the pars orbitalis by a septum interpartes, the orbital portion extending lateralward out into the orbital process of the frontal bone. With the exception of this septum, there are no trabeculae save those indicated by mere ridges to interrupt further the regular outline of the sinus. In monkeys there

Fig. 13.

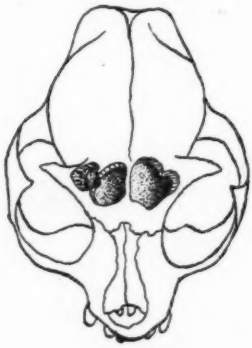

Figure 13.—A preparation of an adult female cat's skull with the sinuses situated in the frontal bones.

s no sinus frontalis present, notwithstanding the well-developed arcus superciliaris. There are, however, a large number of dilated bony cells which occupy a similar position to the sinus frontalis as it is found in the other animals. These rarefied bone cells take the place of the sinus and probably communicate with eachother and the nasal meatus, although injections to prove this point were not made. In horned animals trabeculae are arranged so as to provide not only for the strength of the sinus and the protection of the cerebrum, but also as a reinforcement against stress applied

to the skull through the horns. No gradual increase or decrease in complexity can be followed through the animal series, the sinus seeming to adapt itself in each species toward the architecture of the skull to the end that it affords at the same time lightness and strength.

It has been suggested that the frontal sinuses are being eliminated from the vertebrate skeleton by a process of

Fig. 14.

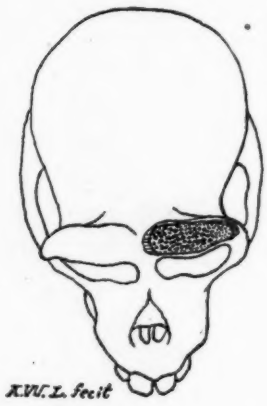

Figure 14.—A preparation of an adult male monkey's skull (species unknown). No sinus exists in the usual form. The arcus superciliaris sin. has been cut and shows the sinus, consisting of dilated pneumatic spaces.

evolution, owing to their occasional absence in otherwise normal human crania, their irregularity in form, extent and capacity, as well as their extreme asymmetry. But a comparison of the sinuses in a series of mammals shows no diminution in their size corresponding to the position of the species in the animal scale. Moreover, the extreme modification presented by the sinuses in monkeys also seems to argue against the plausibility of the suggestion.

Inspection of a comparative table of the relation of brain weight to body weight in the series of mammals compiled by Leurent and Gretiolet reveals the following facts:

For example, in the horse and pig the ratio of brain weight to body weight is respectively 1 to 582 and 1 to 481, that is to say, the proportion is much smaller in the pig than in the horse and yet the former has the largest frontal sinus. It appears from these two instances, which can also be amplified by others in the table, that there is no distinct relation between brain weight in its relation to body weight and the sinus frontalis. This also appears to be the case in reference to the relation between the cranial capacity and the size of skull, for monkeys. which next to man have the highest relative cranial capacity, possess only a modified frontal sinus, as the tissue beneath the well-developed superciliary arches consists simply of dilated pneumatic cells.

XXVII.

NASAL HYDRORRHEA.*

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A considerable literature on this affection has collected since Bosworth called the attention of the profession to it, in a thorough discussion of the subject, and I, in 1889, gave abstracts of the sixteen cases he then found on record. Little has been added to our knowledge since that time, although a number of theories as to its etiology have been offered by different writers.

It seems desirable to establish the entity of cerebro-spinal rhinorrhea. The character of the fluid, alone, would be sufficient to determine such an entity, and to distinguish it from true nasal hydrorrhea. The laboratory tests, as outlined by St. Clair Thomson, are sufficient to determine whether the fluid is cerebro-spinal:

1. Cerebro-spinal fluid is transparent, like water, and has no sediment.
2. It is faintly alkaline, and it may be either tasteless or slightly salty.
3. The specific gravity is 1005 to 1010.
4. It is not viscous and gives no precipitate, (mucin) on addition of acetic acid.

*Read at the meeting of the American Laryngological Association. Atlantic City, N. J., June, 1904.

5. On boiling there is no more than a trace of coagulum of serum globulin or serum albumin.

6. Cold nitric acid gives a precipitate, which disappears on heating and separates on cooling.

7. Saturation with magnesium sulphate should give a precipitate; saturation with sodium chloride should give a precipitate, and ammonium sulphate should be tried if the above fail.

8. The liquid should give a pink, or rose red color, on addition of copper sulphate and excess of caustic potash.

9. It should reduce Fehling's solution.

10. The reducing substances can be separated out by evaporating to dryness an alcoholic extract of the fluid. It is then found as needle-like crystals.

11. An aqueous solution of the residue does not ferment with yeast.

It is generally conceded that this fluid comes from the subarachnoid space and finds its exit through the nasal cavities. Possibly it filters down through the perineural sheaths, or through traumatic openings in the skull, the results of blows, injuries, etc.

Hektoen records a case in which a portion of a meningocele was removed from the nose and cerebral fluid escaped therefrom.

It differs from true nasal hydrorrhea in some of the symptoms, particularly headache, which usually ceases when the discharge is free. Other symptoms, that might be referred to brain pressure, seem to result from the cessation of the nasal flow.

For these reasons the two affections should no longer be classified together, but should be considered as quite distinct in character.

Nasal hydrorrhea is a copious flow of watery fluid from the nasal mucous membrane, either continuing through the entire 24 hours or appearing at certain times during the day, or at irregular intervals, and persisting, sometimes, for long periods of time, possibly unaffected by any treatment whatsoever. It may come from both nares, but is often one sided.

The etiology of the disease has been the subject of much

discussion and there have been various theories advanced. Its neurotic character is generally accepted. The two theories that have received the widest recognition are:

1. It is a neurosis of the tri-facial nerve in the nasal mucosa.

2. It is one of the symptoms of a general neurasthenic condition.

Bosworth believes that there are two classes of cases, in one of which there is an abolition of the function of the tri-facial nerve, followed by a serous exosmosis which takes place without sensation. In a second class of cases, there is an intense irritation of the nasal mucous membrane. Here the integrity of the tri-facial nerve is intact, and there seems to be some disturbance in the sympathetic system, to account for the phenomena. The latter class of cases, he thinks, is closely allied to hay fever, being dependent, largely, upon the neurotic habit, so-called, but differing from hay fever in the absence of a diseased condition of the Schneiderian membrane and the recognized influence of pollen upon it in hay fever. He summarizes his theories, tersely, as follows: "In connection with an intensely neurotic temperament the nasal mucous membrane is rendered sensitive to some obscure atmospheric condition, under the action of which vaso-motor control of the blood vessels, whose specific function is the exosmosis of serum in the normal process of respiration, becomes paralyzed, and then exosmosis takes place to an abnormally large extent."

Kyle, in his study of the subject, contends that in most, if not all cases, the hydrorrhea is a local manifestation of a constitutional condition in which the venous return is impeded, producing a cyanotic condition of the nasal mucous membrane, followed by a leakage of serum into the perivascular spaces, edema, hydropic degeneration of the epithelial cells, etc.

Nikitin considers it a reflex paresis of the vaso-motor nerves of the nasal mucosa, reflex in character, as might be caused by the presence of adenoids, etc., and illustrative of his theory, gives a history of such a case in a girl of eleven years of age, in whom the hydrorrhea followed the removal

of lymphoid tissue in the pharyngeal vault, and which was cured after five months' treatment.

Cathcart reports a case, however, in a girl of nine, in which operation upon the adenoids, twice, and cauterization of the nasal cavities failed to give the slightest relief.

Lawrence had a case, following an attack of edema of renal origin, the flow existing for several years, finally becoming constant, and in which many forms of local treatment were without avail.

Angieras narrates a case following an operation on the sinus maxillaris for empyema, and in which the nares were normal. He thought the condition parotidean in origin.

Molinie cites a unique case in which the development came after an attack of ordinary "grippe" in which the grayish secretion changed to a blue color four or five times during the day. This could not be accounted for. Bacterial examination was negative. It was not hysterical and the color was not introduced from without.

Natier maintains that nasal hydrorrhea is invariably dependent upon a state of general neurasthenia and is exclusively a symptom of that affection, and, in support of his theory, reports 14 cases with a neurasthenic history. He considers that nasal polypi and other intra-nasal pathologic conditions are the effects and not the causes of hydrorrhea, thus accepting an assertion previously made by other observers. He believes the frontal sinus plays an important part in the formation of the liquid, thus accounting for the frontal headaches which some others consider simply plethoric in origin. He also believes that cerebro-spinal rhinorrhea could be proven to be neurasthenic. The hydrorrhea caused by hay fever he thinks entirely distinct, being a special vasomotor rhinitis.

Mayer reports 5 cases, in 4 of which, just preceding the menopause, the nasal hydrorrhea was much augmented during the menstrual period. These cases were all cured.

In this connection a case reported by Laguerriere, last year, is interesting. A woman, 30 years of age, was being treated for pelvic trouble, by means of electricity, and thus was relieved of an obstinate and profuse nasal hydrorrhea.

Litchwitz had a case, in a woman, associated with various

nervous phenomena, which was improved by puncture of the right sinus frontalis, and a year later cure followed the expulsion from the nasal cavity, of a gelatinous mass.

Lermoyez thinks that the disease is much more frequent than his American confreres have believed it to be, and that it is a common ailment among the sufferers from arthritic conditions, of which it is merely an accidental symptom, often alternating with other phenomena of that state, as gout, rheumatism, migraine, etc. Nasal lesions are not constant and are possibly secondary. He further asserts that the fluid is not an exosmosis, but a true secretion, dependent upon abnormal excitation of the trigeminal nerve, and cites as proof that muscarin intoxication produces nasal hydrorrhea. He asserts that there may be vaso-dilator activity but not paralytic phenomena. Pathogenically, the relation between the nasal hydrorrhea and the general systemic condition, and its relation to local or nasal lesions should be carefully studied.

One of the most critical of recent investigators, St. Clair Thomson, in his classical paper on the subject, in 1898, has limited the term "nasal hydrorrhea" to those cases in which no visible pathological changes are found in the nares, and in which the fluid flows from the mucous membrane, without any apparent cause of irritation, in the nose or neighboring parts, and in which the amount of secretion is considerable. He furthermore takes as a standard of the character of the secretion Prof. Halliburton's analysis in his case. This showed:

Water	98.792	Per 100.
Total Solids	1.208	
Proteids, including the mucin	0.260	
Other organic substances	0.163	
Inorganic substances	0.785	

He says that the presence of mucin, the absence of reducing substances, as well as the percentage of proteids and solids differentiate it from cerebro-spinal fluid. He insists that we should eliminate from consideration, in our study of true nasal hydrorrhea, cerebro-spinal rhinorrhea, those cases dependent upon pathologic intra-nasal lesions; those caused by sinus disease or by general nervous affections and those

in which there is not a copious flow, being simply a symptom of such conditions as hay fever, vaso-motor rhinitis, etc.

These are the main theories advanced by investigators. To me, it seems that the neurasthenic character of the disease is fairly well established, and probably the exosmosis of the fluid is due to the irritation of the terminal filiments, in the nasal mucosa, of the tri-facial nerve. The exciting cause is not well established, and is probably due, in many cases, as suggested by several authors, to some obscure atmospheric condition.

The main symptom is the discharge of a clear, limpid fluid from the nose, which examination should determine was not a true nasal secretion. It may be from both nostrils, but often from one only, the quantity varying widely in different cases, from a rather free discharge to a pint or more in 24 hours. The flow may come drop by drop, without intermission in the 24 hours, or may be somewhat periodical in character, possibly ceasing for a time but, in many cases, recurring.

There are other symptoms, as headache, insomnia, cough, sneezing, nervous phenomena, etc., which are by no means constant, but in many cases the discharge is practically the only pronounced symptom of which the patient complains. The nose, on examination, may appear perfectly normal. Kyle noticed a boggy appearance of the mucous membrane of the nasal cavities during the attack, in his case, and there was evidence of a passive congestion and local irritation.

In the treatment some results have been shown by a few, while many report the trial of numerous remedies with little or no influence on the course of the disease.

Linhart's case was cured by the use of Dobell's Solution with a dram of suprarenal extract to the ounce, used as a spray. Similar satisfactory results from suprarenal extract are recorded by Phillips and Berens, the latter using the remedy both locally and internally. Natier recommends a general treatment for the neurasthenic condition, believing local remedies of little avail. Mahu reports a case cured by the use of hot air. Baber used the constant current of electricity and a 20 per cent. solution of menthol as spray and reports his case cured after a few months treatment.

Lermoyez seems to have observed a larger number of

cases than anyone else—42 in number. He deprecates any local treatment, whatever. In 15 of his cases, treated with the usual methods of endo-nasal manipulations, removal of polypi, etc., only two were cured. Of the remaining 27 cases 13 were lost sight of, but he believes some of them were improved. Of the remaining 14 cases, 10 were cured by the use of strychnin and atropin as follows:

Sulphate of Strychnin.....5 milligrams.
" " Atropin.....5 "
Syrup of Orange Peel.....400 grammes.

2 drams of this mixture were taken at breakfast for 10 days; on the following 10 days, 2 doses daily; on the following 10 days, 3 doses, always at meal time. In this way he attacked the hypersecretion with the atropin and the vaso-dilatation with strychnin. He thinks this treatment should always be instituted at first and local treatment should be undertaken only when this had failed.

Other forms of treatment have been referred to by Bosworth, and other authors, among the most important of which are electricity, cauterization, cocain, quinin, etc.

Author's case: Mrs. G. M. F., aged 44, was brought to me, by her physician, Jan. 30, 1904, complaining of a constant dripping of fluid from the right naris, which began December 15th, and had continued, without intermission, since that date, day and night. She had a similar attack, following a cold, 5 years before this; one which lasted for ten days and yielded to local treatment. She was of a decidedly nervous temperament, the condition being much aggravated, at this time, from the persistency of the discharge. Her heredity was good and she had never had a severe illness. She was of robust appearance; and in every respect she was in excellent physical health. She had been under considerable physical and nervous pressure previous to the attack, having nursed a relative through an attack of typhoid fever in October, and was ill in November, and in bed a week, from a streptococcus infection of the throat, which was at first thought to be diphtheritic. Three weeks later she went to St. Paul, to be with a friend whose husband was to undergo a severe operation; she was present at the operation, lasting 1¾ hours, and, for

two days and nights following, was sympathetically consoling the hysterical wife. She became much exhausted physically and nervously. She returned to Chicago, at once, and, on the way to the train, on a very cold day, was suddenly seized with a severe aching, burning and stinging sensation in the right side of the nose, accompanied with sneezing and the symptoms of an acute rhinitis. In three days the watery discharge began.

Status praesens: General health, good. Excellent appetite. Temperature, normal. Pulse, normal. She said she had no headache. The nasal discharge was painless and dripped from the right side, only, on an average of about four large drops a minute when the head was held forward. Her nights were much disturbed, for the secretion continued while she slept, soaking her pillow, and on change of position running into the pharynx and causing choking sensations when it reached the larynx. There was no excoriation of the lip. During the day she wore a napkin around the nose, and constant use of the handkerchief was necessary. The fluid did not stiffen the handkerchief. The nasal cavities were free. They were normal in every respect, save possibly an excessive moisture of the right side, but no site of the discharge of the fluid could be found and the mucous membrane had no swollen or sodden appearance. The throat was also normal. The fluid was examined at the Columbus Laboratory. It was clear, limpid, tasteless, but became clouded on standing; specific gravity 1012; alkaline, sugar absent, albumen absent, cells absent, mucin absent. It was declared purely a serous exudate. A quantity was collected for an hour, during the day, and it was found that 12 ounces were discharged in 24 hours. The patient stated that this was somewhat increased when she was very nervous.

Treatment: She was given a pill of zinc phospide, nux vomica and quinin, with benefit to her nervous condition and bromidia to quiet the restlessness at night. She had already had treatment at the hands of other physicians, by electricity, bromides, belladonna and varied local applications, without benefit. I used, at first, 5 per cent. protargol, then 50 per cent arygrol, to the nares, and gave her a spray of adrenals 1-5000 and Dobell's Solution to use at home. I

tried, in succession, cocain, adrenalin, cauterization, zinc salts, strong applications of menthol in albolene, and internally, belladonna, aconitine, etc., without the slightest benefit. Finally, I used a solution of argentum nitrate in small quantity, 60 grains to the ounce, and over a portion only of the nasal cavity. This caused some pain and a little headache, but immediately following there was a cessation of the dripping for a few moments. On February 20th, I swabbed the the nasal cavity first with 1-1000 suprarenalin solution thoroughly, and applied the 60 grain solution of nitrate of silver to the entire accessible parts of the nasal cavity The pain was severe and she went home and went to bed with a violent headache, caused by the treatment. On February 25th, the headache ceased, the discharge began to decrease rapidly, and in three or four days ceased entirely, and has not returned.

In this case, it seems to me, we have a true nasal hydrorrhea. The character of the fluid differentiates it from cerebro-spinal rhinorrhea, and headache was not present. Thomson gives as among the evidences of the discharge being cerebro-spinal, that it is usually one sided, continues during the whole 24 hours, does not stiffen the handkerchief, and the nose presents no pathologic changes. Notwithstanding the presence of these conditions, in my case, the inception of the disease, with intense nasal irritation, with the general nervous symptoms and absence of any symptomatic evidence of the fluid coming from without the nasal cavity, I am satisfied that there was first a marked irritation of the terminal fibres of the vaso-motor nerves in the nasal mucosa, followed by paresis, and an exosmosis of fluid through such loss of nervous control.

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XXVIII.

THE PATHOLOGIC AND PATHOLOGICO-ANATOMIC INVESTIGATION OF THE DISEASED MIDDLE EAR DURING THE LAST DECADE, AND THE CHANGE IN OUR VIEWS OF ITS THERAPY CAUSED THEREBY.

By Dr. K. Grunert.

HALLE A. S.

Translated By Clarence Loeb, A. M., M. D.
ST LOUIS, MO.

While I was busy during the last decade with the preparation of the Year Book on the Middle Ear as a part of the large work of Lubarsch and Ostertag (Ergebnisse der allgemeinen Pathologie and pathologischen Anatomie der Menschen und der Thiere, J. F. Bergmann, Wiesbaden); I kept in mind the thought of collecting from the Year Book the most important facts, and of putting them together in a readable form for our circle of readers, in order to acquire thereby a review of the most important advances in the pathologic anatomy of the middle ear during the last decade. Inasmuch as it is the object of this journal to serve the interests of theoretical as well as of practical otology, it was necessary in places to draw practical conclusions from theoretical findings; in other words, to see how far theoretical results could be made practicable, and also to settle the question as to what changes they necessitated in our therapy.

Although theoretical investigation and practice must go hand in hand and the latter must follow the former, this following must be no blind one, but rather one of careful test-

ing and judgment. We must always bear in mind that our theoretical views often undergo daily variation. It would hardly be proper for the practicing physician to be always prepared to draw the therapeutical sequence blindly from such changeable theoretical views. As much as life advances in theoretical investigations, just so critically scrutinizing must be the advance of the physician. Practice should always observe a healthy conservatism, which, in consequence of the constant relationship between theory and practice should hold it equally far from standing still as from too eager discarding of well tested remedies.

In last year's Congress at Karlsbad, I took occasion to show that our specialty had not always avoided this reef and the following arguments support the correctness of this statement.

GENERAL REMARKS.

An intelligent division of labor between otologists and bacteriologists together with the aid of pathologic anatomy, in investigating the relation of pathogenic microorganism to diseases of the middle ear has resulted during the last decade in determining the organisms that cause the inflammation in the ear, and those that are present only as a secondary infection. This was possible in spite of the difficulties of the technique, and in spite of the connection of the diseased middle ear with the external world by two canals, one of which, the Eustachian tube, cannot be closed against secondary infections.

As the foundation for the correct interpretation of the relation between microorganisms and diseases of the middle ear, it was first necessary to determine whether or not bacteria are ever present in the normal middle ear. This question was answered in the negative as far as dogs and rabbits were concerned by Lannois[1] in the year 1896, and by Preysing in 1899 in regard to men[2]. These investigations of Preysing have been lately confirmed by Hasslauer[3].

Although the cause of this immunity of the normal middle ear to bacteria has been regarded as due to a bactericidal property of its mucous membrane, it is rather the protection

afforded by the movement of the cilia of the epithelium lining the Eustachian tube toward the pharynx, by means of which the ear is well able to protect itself against the invasion of bacteria from the nasopharynx. Every injury to the tubal epithelium leading to a retarding or cessation of the movements of the cilia, opens a passage way to the middle ear. With this in mind, we can understand the tendency of febrile infectious diseases to result in diseases of the ear, in so far as the ciliated epithelium of the tube is injured in its biological properties by the high fever itself, and the toxines of the disease. No special proof is needed, that the resistance which the tube normally affords to the passage of germs into the ear can be mechanically overcome, and thus artificial otitis can be formed. To this group belong the otitides that are caused by violently blowing the nose, and the forcible entrance of infected nasal secretion into the middle ear, and also those following the use of the nasal douche, most bath otitides, and those caused by the use of snuff.

Although all investigators are agreed that the usual gateway of infection of the middle ear is the Eustachian tube, it is probab e on the other hand that certain forms of otitis owe their origin to a hematogenous infection. It is just the last decade that has enriched our knowledge of this phase.

Thus Barnick[4] claims that acute tuberculosis of the ear owes its origin almost exclusively to a great overflow of tubercle bacilli into the blood, which in numerous other organs causes a rapid developement of miliary tubercles. But he also claims a hematogenous infection is the cause in a small number of chronic aural tubercul ses especially in scrofulous children, in whom, following a rupture of a cheesy focus, poor in bacilli, the infection-carrier is distributed very sparcely. Whether the first seat of the specific infectious organism is in the mucous membrane lining the middle ear or the temporal spongiosa, can scarcely be proven with certainty in many cases, since the disease often passes immediately over from one tissue to the other.

Furthermore, Levin[5] has shown the probability that the majority of the cases of acute otitis, accompaning pharyngeal diphtheria and running the usual course of an otitis media, are the result of local manifestation of diphtherial poison cir-

culating in the blood, and not of a propagation of the pharyngeal process to the middle ear by way of the tube.

We are of course at present unable to solve the question of the hematogenous infection of otitis complicating those infectious diseases whose specific germ we do not know, e. g., measles and scarlatina

In those malignant forms of scarlatinal otitis characterised by rapid destruction of tissue, in which Leutert[6] always found streptococci, only, in the diseased ear, the often simultaneous appearance of the disease in all parts of the ear (panotitis) speaks for a hematogenous infection of the ear. Although it has been known for some time that the streptococcus has great influence in bringing about scarlatinal otitis, Leutert thinks he has proven that this organism is specific for it. He says "that the streptococcus is the germ that gives to scarlatina the picture of a severe disease, while the so-called specific germ of scarlatina seems to have no relation to the severe general infection and suppuration."

The statement of Zeroni, that the infectious osteomyelitis accompanying otitis arises by way of hematogenous infection, is much less certain than the probability of a hematogenous infection as the cause of certain forms of inflammation of the ear in tuberculosis and pharyngeal diphtheria.

In opposition to the surgeons, who regard an osteomyelitis of the temporal bone as an everyday occurrence, we must insist that up to now not a single case is known in otologic litarature which proves beyond doubt the presence of osteomyelitis in the temporal bone. Even the important cases of Brieger[8] and Moure[9], where there was an empyema of the mastoid not preceded by an otitis media, allow the possibility that there was no true osteomyelitis but only a disease of the mastoid mucous membrane on a hematogenic basis.

A very remarkable case reported by Zeroni from the surgical clinic of the University of Halle(Geh.-Rat. v. Bramann) teaches how careful one must be with the diagnosis of osteomyelitis of the temporal bone even when there is a general osteomyelitis, and a diseased focus in the temporal bone without a previous inflammation of the middle ear.

It was the temporal bone of a 12 year old boy who had died of a general osteomyelitis.

The microscopic examination showed "all nutrient canals of the bone were normal and free from inflammatory signs. There was found, however, larger or smaller spaces in the bone of the superior meatal wall filled with leucocytes of widely varying sizes, and frequent variations in the shape of the nuclei. All of the spaces were lined with cubical epithelium which indeed was not present everywhere, but was at least demonstrable in some place in every space. The epithelium was likewise infiltrated with leucocytes, and the underlying connective tissue was greatly hypertrophied. Here also were found leucocytes, but only in layers lying immediately under the epithelium. Similar pictures were to be found at other places in the temporal bone, especially in the cells lying around the antrum, Only the cells of the tip were free and entirely normal. The mucous membrane of the middle ear also showed no inflammatory changes."

There was, then, in this case an inflammation of the temporal bone accompanying a severe infectious osteomyelitis, but "it was an inflammation of the mucous membrane lining the mastoid cells, which was independent of disease of the middle ear." Zeroni believes that it was a disease of the mucous membrane on the basis of a hematogenous infection; he justly remarks that if this disease had not been seen in a beginning stage, but later, when the inflammation had subsequently attacked the medullary spaces of the spongy bone and the Haversian canals, it would have been possible, considering the general cause, to regard a primary inflammation of the medullary spaces of the bone as the starting point of the process.

In regard to the individual microorganisms found in the middle ear, the reader of the numerous works that have appeared in the last decade will often find a contradiction in the finding of individual investigators. The explanation of this lies not in a failure of observation due to scanty bacteriologic training, nor in generalization based on conclusions drawn from insufficient material, but rather in the difficulty of bacteriologic investigation in this very locality. The difficulty of obtaining the material for examination, pure from the deep-lying middle ear, the connection of the foci of disease with the external world by two ways that cannot be closed against

secondary infection, viz., the external meatus and the Eustachian tube, and the tendency of the inflamed middle ear to mixed infection, explained on these anatomic grounds, show why it is that one investigator considers one of the microorganisms found in the suppurating middle ear to be the specific germ, while another ascribes to it a secondary role.

It is certain that acute otitis media is especially a monobacterial disease, while a polybacterial fauna is always found in chronic suppuration.

If, in studying the relationship of the different organisms to the production of otitis media, we select the investigation of Leutert (l. c.) from the numerous works dealing with the subject, it is because his material for investigation seems to be the freeest from contamination. While the other investigators took their material, more or less pure, from the middle ear, and thus, by possible touching of the meatus, opened a way for a source of error in their experiments, Leutert took his direct and pure out of the collection of pus in the mastoid that he had just opened. In 63 mastoid abscesses following acute suppuration of the ear, he found streptococus 38 times in pure culture, pneumococcus 11 times in pure culture, staphylococcus 5 times in pure culture, pure tuberculosis 2 times, and in the other cases mixed infection, impurities, or doubtful results. The immense preponderance of streptococci in acute empyema, he ascribes to the freqency of streptococcus otitis, which Green[10] also showed lately in a collection of 73 cases, in which he examined the first drop of pus that followed a paracentesis. That Leutert observed the staphylococcus less frequently than the other authors, finds a very plausible explanation in the circumstance that their material often came in contact with the meatus.

In contrast to the relative malignancy of the streptococcus, which is manifested in the fact that intracranial complication lie especially in the domain of this microorganism, Leutert and the other investigators (Zaufal, Netter, Maggiora, Gradenigo, Vernieull, etc.) claim for the pneumococcus a relative nonmalignancy in its character as a cause of otitis. He agrees with Maggiora and Gradenigo, who partially explain this lack of malignancy by the fact that the pneumococcus is always

found in the aural pus in a state of weakened virulence, and he also found this same condition in the oral cavity, the original source of this infection. The preponderance of the pneumococcus in the empyemas located in the tip of the mastoid, as well as the rarity of pneumococcus infection in the young has so far not been explained.

Of great interest is the circumstance first noted by Zaufal but emphasized by Leutert, that the pneumococcus usually makes a distinct clinical picture in the otitis caused by it. According to Leutert, usually, though not invariably, the otitis caused by the pneumococcus is distinguished from that caused by the streptococcus by three points: (1) the acute processes in the middle ear pass away quicker in the pneumococcus form; (2) the pneumococcus has a greater tendency to spread beyond its original infected area, than the streptococcus (more frequently epidural abscess); (3) After cessation of the acute processses in the middle ear, the pneumococcus infection often remains for a time latent before it again becomes acute in the mastoid.

Leutert considers the importance of the staphylococcus in the etiology of acute mastoiditis as so minimal, that he even leaves the possibilty open that a secondary infection had already taken place in the few cases of acute empyema where the staphylococcus was present.

Other authors, such as Pes and Gradenigo[12], and later Green (l. c.), explain the chronicity of an acute otitis media on the ground of the individual anatomic conditions as well as pathologico-anatomical factors, a point of view which finds a certain support in the connection recently established by Roaldes[13] between certain anatomic conditions and predisposition to middle ear inflammation. Leutert, however, as well as some of his predecessors, considers the secondary infection with staphylococci, especially staphylococcus albus, and with saprophytes to be the chief cause of the chronicity of the middle ear suppuration. At any rate, according to him, these secondary invaders enter from the naso-pharynx through the Eustachian tube and not, as Lermoyez and Helme[14] claimed, through the perforated drum. Leutert did not assert this from a purely bacteriologic standpoint, but rather from a mixed one, since he re-

cognized in the mentioned pathologico-anatomic factors an influence in bringing about the chronicity of the ear suppuration, insofar as they lengthenened the process of healing, and thus created favorable circumstances for the entrance and development of staphylococci and saprophytes.

Hasslaur (l. c.) thinks upon the basis of his investigations, that he could dispute the view of Leutert, that it was chiefly the secondary infection with staphylococci which brought about the chronicity of the suppuration, but does not present convincing proof.

Only the most important of the facts concerning the bacteriology of the diseased middle ear, that have been proven in the last decade, will be mentioned. Kossel[15] was the first to demonstrate the presence of Loeffler's diphtheria bacillus in the pus of an acute otitis media, in a child with diphtheria.

Stern[16] showed that the bacterium coli is found in a suppurating ear, while Leuter found it in an otogenic brain abscess. Zaufal[17] deserves the credit of first showing the pathogenicity of actinomyces in the middle ear; he thought that in his case (suppuration with granulations, in which the the mycelia were found) the middle ear was the primary source of this disease, and the actinomycotic foci under the deep musculature of the neck and under the cranial cavity had arisen by extension of the disease along the incisura mastoidea.

Cozzolino[18] thoroughly examined a case of pseudo-actinomycosis of the ear, which clinically and microscopically was very similar to a true actinomycosis (granules in the pus, muco-sanguinous character of the exudate, etc.). The cause of this disease, the bacillus filamentosus, up to that time unknown, according to Cozzolino, does not belong to the ray-fungi family, and is also to be differentiated from the bacillus pseudoactinomycosis of Sawtchenk and of Krassnobajew, which belongs to the schizomycetes. In contrast to the latter, the bacillus filamentosus is aerobic, stains according to Gram, forms spores, is very resistent to heating and drying, and shows many of the characteristics found by Kruse in the family of the hay bacillus. He laid the piece of tissue obtained at the autopsy for 3 months in 10 per cent.

formalin, which he frequently changed, and at the end of that time made cultures from the pus in the meatus and cerebellum on bouillon and agar. Out of 12 inoculations, he obtained 8 times pure cultures of the germ, although the development of the cultures was slower and scantier.

The demonstration of the bacterium aerogenes (Escherich) in the middle ear of a case of Scheibe[19] that died of purulent meningitis, in which the same bacterium was also found in the meningitic pus, is worthy of mention.

In a serous middle ear exudate obtained by paracentesis of the drum, Barrago-Ciarelli[20] found a blastomycetes very similar to the blastomycetes lithogenes, which he could grow in pure cultures on bouillon and gelatine.

Finally, Schilling[21] proved the pseudodiphtheric bacillus to be the cause of an otitis in a case of genuine croupous otitis media in a patient 24 years old. Anatomically and clinically, the case was characterized by the protrusion of white membranes out of the paracentesis opening in the drum, which were shown by Weigert's fibrin stain to be a fine network of fibrin fibres with granular and globular fibrin. In its further course, the suppuration took on more the character of an ordinary middle ear suppuration. The bacteriologic examination showed diplococci and diphtheria-like rods. "The above mentioned diphtheria-like rods can be regarded only as pseudodiphtheric baccilli, on account of their peculiar properties (richer growth on agar than on blood-serum. notched border of the colonies, not very numerous granular staining, and a pathogenicity for guineapigs). As at the section of the animals experimented on, none of the changes characteristic for diphtheria were found, but on the contrary the presence of pneumonia diplococci was demonstrated, it is probable that the death of the animals was caused by them." At the beginning of the disease, which was characterised by the appearance of white fibrinous membranes, the pseudodiphtheric bacilli were most prominent, but were later supplanted by the ever-increasing diplococci, as the case took on the character of an ordinary otitis media. This fact gives a certain support to the supposition that the original disease is to be ascribed to the pseudodiphtheria bacilli.

We lately saw in the Halle Ear Clinic a case characterised by a similar clinical course, in which, however, the bacteriologic examination, carried out at the Royal Hygienic Institute, showed the presence of the true diphtheria bacillus, in addition to staphylo-and streptococci.

WHICH OF THE ABOVE FACTS ESTABLISHED DURING THE LAST DECADE CONCERNING THE FAUNA OF THE MIDDLE EAR ARE APPLICABLE IN PRACTICAL MEDICINE, ESPECIALLY OTOLOGY?

In the first place, in regard to the prophylaxis, the fact must be acknowledged that the tuba Eustachii is the usual port of entrance of pathogenic bacteria into the ear, and that a weakness of the movement of the tubal cilia opens the way for bacteria to pass from the nasopharynx into the ear.

From this it is evident that the greatest care must be taken to clean out the nasopharynx, as far as that is possible considering the anatomical relations. This is especially necessary in diseases of the upper air passages, in which infectious secretions are present in the nose and naso-pharynx in unusually large amount.

The method of cleansing to be chosen depends on the case and on the character of the secretion found in the nasopharynx. In the majority of cases, it will be sufficient to use a v. Troeltsch nasal douche with a neutral 0.75 per cent. salt solution, or a weak boric acid solution, together with rinsing the mouth. Especially in acute infectious diseases, it is necessary by this cleansing to protect the patient from the imminent danger of an otitis, the more so since here on the one side, the normal protection which the ear possesses in the movement of the cilia of the tube is more or less lost, and on the other hand, the bacterial fauna of the mouth is better developed on account of the lessened masticatory movements of the mouth, resulting in a diminution of the normal power of self-cleansing. That carious teeth should be given the attention that they deserve is self understood.

That by these prophylactic measures, it is possib'e to prevent an otitis is proven by numerous observations at the

sick-bed. I myself during my period as a first-year physician passed through a typhoid epidemic, in which not one of 30 patients had an otitis, thanks to the most careful observation of this prophylaxis.

As far as the artificial otitides are concerned, i. e., those following nasal douching, bathing, and use of snuff, the prophylaxis is given by the disease.

If we get a well-developed case of otitis media acuta suppurativa we must use our best endeavors to prevent it from going over into the chronic form. The basis for the prophylactic effort is given by the fact that the tendency to chronicity of an acute otitis makes its appearance when there is a secondary infection of the ear, especially with staphylococci and saprophytes. This knowledge forces us not only to provide for a free drainage of the pus but also to protect the ear against secondary infection. In regard to one way by which this can take place, the external meatus, we attempt to solve this by carefully cleansing, at every change of dressing, the vicinity of the ear and especially the numerous recesses of the auricle, those breeding places of saprophytes, and then guard the ear by an aseptic dressing. As to the other way by which secondary infection can take place, the Eustachian tube, our prophylactic measures agree with those against the origin of otitis in general.

The standpoint of preventing a secondary infection will cause us to limit the paracentesis to those cases where it is actually indicated (i. e., where there is a purulent exudate behind the drum), since a paracentesis opens the way for an infection of the middle ear from without.

Furthermore, in relation to the prognosis, the above progress of our knowledge of the bacteriology of the ear allows us to draw certain practical conclusions, whose possession can be obtained by the bacteriologic examination of the pus in each case of acute otitis media purulenta. If we recognize a suppuration of the ear as a pneumococcal one, the prognosis in regard to its length, course and termination is in general, much more favorable than if it were a malignant streptococcal infection. If it is a monobacterial infection, the above turns out, in general, much better than if it is a polybacterial.

Although the pneumococcal otitides have a relatively harmless character, still the fact that in this form, after a rapid disappearance of the otitis media, the infection can appear in the mastoid or even the cranial cavity, after a period of latency, makes it necessary for us to watch for some time cases of otitis media due to pneumococci which result in a rapid cure, and to acquaint the patient with the possibility of a subsequent return of the infection in the mastoid.

Our knowledge of the otitis media neonatorum, and of the otitis of nurslings has become broadened and deepened in the last decade. Although v. Tröltsch a decade before had drawn our attention to the significance of the frequent otitis media in the earliest childhood, it remained for a number of diligent workers of the last decade to shed more light on the general and special pathological features of this disease. Aurists and specialists in bacteriology and pathology shared equally in this fruitful work.

The otitis of the new-born and that of nurslings were not always sharply differentiated, and yet this distinction is an important necessity, since it alone gives the solution to the question whether or not there is an otitis media of the new-born whose origin must of course be referred back to the period of intrauterine life.

This question must be answered in the affirmative since the investigations of Aschoff.[22] He was able to put an end to the discussion waged since the 60s as to whether the pus so often found in the middle ear of new-born children was physiologic and perhaps had some relationship to the disappearance of embryonal gelatinous tissue or whether it was to be considered as the end product of inflammation. He came to the following conclusions, based on the microscopic examinations of the contents and mucous membrane of the middle ear of 85 new-born children:

(1) Unless there has been a complete entrance of air after extra-uterine breathing, a fluid is found varying from clear watery-like to that of muco-purulent. In a large number of cases the contents were mixed with constituents of the amniotic fluid. (2) The formation of pus in the middle ear of the new-born is no physiologic process necessary for the formation of the middle ear, since it is not found in all

fetuses, but is rather the result of a very early (from the fourth month of fetal life) entrance of the constituents of the amniotic fluid into the middle ear. This reaches the highest degree in the entrance of large masses of vernix and meconium, which can result in the greatest amount of pus formation, viz., a complete filling out of the middle ear with pus. (3) The mucous membrane of the middle ear of the newborn shows a varied degree of cellular infiltration, which increases corresponding to the amount of irritation of the larger or smaller masses of the constituents of the amniotic fluid—the meconium and vernix—but never reaches the degree that is found in a severe case of infectious otitis media. It may, however, closely resemble the picture of a mild inflammation, if it is supplemented by hyperemia and hemorrhages caused by suffocation. (4) The disappearance of embryonal gelatinous tissue takes place intrauterine, according to developmental laws unknown to us, but not as the result of coarse, mechanical influences, as Wreden, Wendt, etc., thought; the width of the cavity allows absolutely no inference to be drawn as to a pre- or postnatal breathing.

Although, as Wendt has already shown, the otitis media neonatorum has been proven to be a foreign-body otitis, the question of the pathogenesis of the otitis of nurslings and of early childhood has not yet been sufficiently cleared up. It can, however, be seen from the works dealing with the question that this disease is to be considered as a single, sharply defined disease of the middle ear neither from a pathologico-anatomic standpoint, nor from its etiology. The views of the individual authors disagree partially according as each investigator starts out more from a pathologic or anatomic standpoint. There are even those who claim that the exudate found in the middle ear of 80 to 91 per cent. of all children during the first four years of life is often of preagonal or agonal origin. Berthold[23] especially, takes this position and says that in the condition of preagonal and agonal somnolence the mucus of the naso-pharynx gets into the middle ear. The micro-organisms entering with the mucus develop and cause an otitis, which could be called an otitis media infantum moribundorum. Even more

skeptical as to the frequency of otitis media in early childhood is George Paul Veillard[24], who on the ground of the findings at 80 postmortems, comes to the conclusion, in many cases, of a "simulating-otitis." This author found in 30 per cent. of his case pus in an otherwise normal middle ear, all cases where the pus had entered from the diseased naso-pharynx into the healthy middle ear, and the mucous membrane in only a few times had reacted by injection of its vessels. In the cases, however, of otitis and not "simullating-otitis," the author found an involvement of the bony walls of the middle ear, which has lately been confirmed by Politzer[25], who demonstrated an accumulation of round cells in the medullary spaces of the spongiosa.

Although the investigations of Veillard (l. c.) show that the otitis media in early childhood is often no true otitis, and although the view of Berthold, that in many cases we have only to do with a preagonal or agonal incident, has much in its favor, the generally severe character of the disease has been well shown by the work of Hartmann [26] and [26]a, Geppert[27], Barth[28], Ponfick[29], Heermann[30], and Weiss.[31]

The results of the bacteriologic investigations of the individual authors do not agree. The results of Kossel, who examined bacteriologically 88 cases in the Berlin Institute for Infectious Diseases, differ considerably from the corresponding findings of the earlier investigators, Rasch, Netter, Gradenigo and Penzo. The consensus of the findings show beyond doubt that the otitis media acuta of nurslings is no etiologically circumscribed disease, but that widely different microorganisms play a causal role in its orgin, as in the pathogenesis of otitis media acuta in general.

Though the authors just mentioned lay a too one-sided stress on the bacteriologic standpoint in regard to the origin of the nursling's otitis, and though others lay a too one-sided emphasis on the anatomical susbtratum, Weiss[31] has skilfully avoided this one-sided view by combining the bacteriologic and the anatomic examination of the middle ear. He examined the middle ear of 28 nurslings, mostly pedatrophic, who had died from diseases of the respiratory and digestive organs. The most important result of his histologic investigation is that it is always possible to demonstrate

the presence of the embryonal character of the nursling's mucous membrane of the middle ear. "Even where the densest infiltration makes the recognition of this structure of the mucous membrane almost impossible, the layers lying deeper and near to the bone, which have been protected from the extreme cellular infiltration, show plainly the stellate cellular network in the homogeneous ground substance." Weiss recognizes in this deviation from the character of the mucous membrane of the adult a "factor of probably etiologic value in explaining the enormous frequency of middle ear disease in nursing babies and its very special form." The acceptance of a tissue predisposition, which possibly consists in a greater sensitiveness and vulnerability of the characteristic mucous membrane of that period of life, makes us understand better the peculiarities of the otitis of nurslings. The bacteriologic investigations of Weiss confirm the statements of earlier investigators—Kossel, etc.—that the otitis of nurslings is caused by the known forms of germs; the infection can take place either via the Eustachian tube or the blood. Note should be taken of the frequent finding of the diplococcus pneumoniae, 42 per cent., to which Weiss acribes the relative benignancy of the otitis of nurslings, as well as the slight tendency to rupture of the exudate through the drum.

Although, according to the above investigations, the otitis of nurslings is characterized as a genuine local disease of the ear, Heermann[39] considers it only as an accompanying symptom (otitis concomitans) a result of the marasmus caused by other diseases: "The concomitant otitis very seldom, only when it reaches an intense degree of inflammation, leads to spontaneous rupture of the drum, the genuine almost always cause a perforation, or by the severity of its symptoms calls for instrumental interference. Now since the one leads to the same complications as the other, empyema of the mastoid process, meningit's, otitis pyemia and sepsis, their differentiation is impossible, if only the end of the process is regarded." This view of Heermann seems to rest upon confusion of effect and cause. Much bette: grounded is the view of Ponfick[29] with respect to the pathologic relation of the otitis of infancy, who regards the inflamed middle ear as

often the result of a secondary disease Ponfick, who examined 100 children up to 4 years of age, of whom almost three-fourths belonged to the first year, found that among 6 children dying of acute, non-infectious processes (congenital heart failure, etc.) 5 had otitis media purulenta, which speaks against the view of Heermann. Of 65 dying of acute infectious dermatitides (furunculosis, erysipelas) diphtheria, scarlatina, inflammatory thickenings of the lungs, purulent meningitis and gastroenteritis infantum, only 7 had a normal middle ear, while 58 had severe inflammation, the exudate being occasionally serous, but usually purulent (eight times unilateral, 50 times bilateral). In speaking of the relation between gastroenteritis and pneumonic foci, Ponfick claims that both respiratory and intestinal disturbances can be induced either simultaneously or immediately subsequently by infection from a general focus, viz., the ear. In10 other cases, where the autopsy was negative except for a moderate enlargement of the spleen, the middle ear was the only organ showing considerable changes. These investigations of Ponfick show that the life of the nursling can be as much threatened by an otitis media alone, as by inflammation of the air passages. The fatal end need not be caused by the appearance of an intracranial complication, but comes rather from the entrance of the toxins of the otitic process into the nutrition. According to Ponfick, the otitis is not only a local but also a general affection, and not as Heermann claims, the accompaniment of a general disease. That the otitis is not a focal but a general disease is confirmed by the feverish state, the frequent enlargement of the spleen, the degenerative processes noted by Ponfick, especially in the kidneys and liver, the intestinal disturbances, etc. Among 19 children dying of chronic infectious diseases (16 tuberculosis, 3 syphilis congenita) the drum was found only once uninvolved.

The results of the investigations of Hartmann[26a] on the influence of the otitis media of nurslings on the nutrition confirm Ponfick's view as to the general pathologic relationship of otitis media in early childhood. His conclusions are as follows:

1. The otitis media of nurslings may be accompanied by

nutritive disturbances, which find their expression in disturbed digestion and loss of weight.

2. With the discharge of the pus, by paracentesis, the digestion in these cases may again return to the normal, and the loss be followed by a gain in weight.

3. Increase of temperature which appears in the course of an intestinal disease in nurslings may be caused by an otitis media.

4. In all intestinal affections of nurslings accompanied by increase of temperature and loss of weight, the ear should be examined for the traces of inflammation.

THE PRACTICAL APPLICATION OF THE FACTS CONCERNING OTITIS MEDIA OF INFANCY ARE SELF-EVIDENT.

It is not necessary to state that the aurist must treat this form of otitis with the same care as every otitis of later childhood and adult life. This otitis has a special place in therapy since the drum shows a slight tendency to rupture spontaneously—less the result of the so-called "relative benignancy" of the otitis than of the great resistance of the tympanic tissue of the child—and since, in spite of the purulent contents of the middle ear, the local symptoms are frequently entirely masked by the general. The cause of this is perhaps that the wide tube of the child allows a freer exit of the secretion toward the throat, and therefore the retention of pus, with its severe local symptoms, occurs less readily.

Although the lack of retention makes the danger of intracranial complications less probable, the flow of the pus through the tube into the throat increases the danger of secondary disease of the intestinal tract, with the resulting severe disturbances of nutrition.

This danger puts the paracentesis needle in our hands much more frequently than is otherwise the case. Even when there is no fulminating symptom present, it is our duty to bring about the flow of the pus outward, and thus prevent its flowing into the tube.

The general practitioner should lay to heart the lesson taught by the above facts, viz.: that every nursling with general symptoms, with intestinal disease, with catarrhal disease of the lungs, with diseases of the nutrition, etc.,

should have its ear carefuily examined, even when no symptom on the part of this organ seems to indicate an examination of the same.

But above all, the above general pathologic relation of the otitis media in early childhood makes it neccssary for every pediatrist to be thoroughly conversant with the examination of the ear of children. If he lacks this abilit·, he is frequently in danger of diagnostic error and of completely overlooking the facts, resulting in the probability of failure of his therapy.

Anyone who believes that otology is a narrow specialty, having few points in common with general medicine, can easily learn differently from the numerous works that have appeared in the last decade, which have demonstrated the numerous points in common of general medicine and otology. I have used the words "points in common" and should like to insist that in the last decade not only has the pathologic anatomy of the ear received great amplification on account of its close relation to general medicine, but the latter, also, has been enriched and advanced by otologic investigations. I need only speak of otogenous pyemia which has radically changed our views of the pathogenesis of pyemia, in general.

The works of the last decade that deal with the relation of diseases of the middle ear to other general and local diseases are so numerous that we must pass over those that contain only a theoretical exposition of our science.

A complete change has been wrought in our views as to the relation between diabetes and diseases of the middle ear.

Although formerly it was believed that the middle ear disease in diabetes was specific, and though, above all Kuhn, Körner, Walb, and Wolff taught that in diabetes the mastoiditis was primary and the otitis media secondary, Davidson[32] insisted that the diabetes was only a predisposing factor in bringing about the disease of the ear. Brieger[33] took the same standpoint, since he ascribed to the diabetes no causal role in bringing about the otitis media, and credited it with influencing an intercurrent otitis media only in so far that he accused it of intensifying the process, and of perhaps favoring the development of the wide-spread necrosis. Eu-

lenstein[34] has dealt even more thoroughly with the question of a specific "otitis diabetica," by critically reviewing the 46 cases reported in literature. "If we examine the material before us objectively and onsider that the picture of the otitis diabetica s. necroticans was formulated at a time when there were only a few observations, we must say that this picture owes its origin to a generalization of a few observations which have not been confirmed by subsequent investigations, and the view of certain authors is not correct; wc will come closer to the actual condition if we speak of an otitis media of diabetics in the sense, that under conditions still to be determined an otitis media purulenta in a diabetic patient may easily cause severe complications in the mastoid or middle ear, which are usually manifested by a rapid extension of the process and extensive carious destruction of the bony tissue, and occasionally by actual necrosis of the bone." The "conditions still to be determined" of Eulenstein are the following: 1. The anatomic construction of the mastoid (pneumatic, etc.). 2. The favorable culture media for the most varied germs, brought about by the diabetes. 3. Arteriosclerosis with its consequences in tissue necrosis. 4. The severity of the infection.

I have shown[35] that not only does diabetes mellitus give a distinct anatomic and clinical character to middle ear disease but also that ear disease may elicit the chief symptom of deranged metabolism, viz., glycosuria. Although both cases, observed in Schwartze's clinic, are cases of otitic intracranial complication, still mention may be made of this phenomenon at this place on account of the interesting general pathologic relation of the glycosuria. The few cases heretofore reported in literature dealing with the causal relationship between otitis and glycosuria will not stand a critical examination. The two following conditions must be fulfilled in showing a causal relationship between otologic sequelae and sugar in the urine: first the proof that there was no sugar in the urine before brain disease, and second the proof that the glycosuria that appeared during the otitic complication disappeared completely after the brain affection healed. Both conditions are fulfilled by the two

cases observed by us in the Halle ear clinic. In the first patient, suffering with chronic middle ear suppuration, the urine on entrance was free from sugar, the patient eating a mixed diet. During the exacerbation of the cerebral symptoms, already present at his entrance, there developed a stubborn glycosuria. This entirely disappeared after a large mass of exudate had been removed from the cranial cavity, by an operation. It cannot be settled whether the glycosuria in this case is to be regarded as a result of a sympathetic exudate into the ventricles, caused by an intraventricular increase of pressure, especially in the 4th ventricle, since clinical experience teaches that glycosuria does not always appear in every case of such an otogenic, intraventricular increase of pressure. The second case was that of a 47 year old man, with a right-sided acute middle ear suppuration, and an extradural abscess the size of a cherry, in the posterior cranial fossa. The urine of the patient on entrance was likewise free from sugar, on a mixed diet. Two days later, the same patient, on the same diet showed sugar in the urine, which completely disappeared after the abscess was emptied by an operation. On account of the small size of the extra-dural pus focus, we do not believe that any considerable increase of pressure in the cranial cavity can be assumed as the cause of the glycosuria. We think the most probable explanation is that the glycosuria was caused by tonic influences and cite the analogy of the glycosuria observed in phlegmonous acne and septic processes, which are designated as toxic.

While according to the above, our knowledge of the relation between diabetic anomalies of metabolism and otitis has received a considerable accession by the labors of the last decade, much has been written concerning the relation of the uratic diathesis to the ear but nothing had heretofore been definitely proven. It was therefore a great service of F. Alt[36] when by means of experiments in metabolism he investigated thoroughly in otosclerotic patients the theory held especially by English authors, of a causal relationship between sclerosis and uratic diathesis. The results of his investigations in no way affirmed such causal relationship,

so that we can no longer regard gouty anomolies of metabolism as an etiologic factor in sclerosis.

As to the relationship of middle ear diseases to other general diseases, Schwabach[37] has added another case to the single one of Habermann, of disease of the ear in pernicious anemia, in which hemorrhages were found in the sound perceiving apparatus. The histologic examination of Schwabach's case showed that the sound perceiving apparatus was normal, while the middle ear showed wide-spread changes. They consisted of more or less extensive extravasations of blood, especially on the floor of the middle ear, in the niche of the oval window, in the drum (subepidermoidal, hemorrhagic vesicles), and in the cells of the mastoid. In addition, there were signs of a secondary inflammation, probably caused by infection from the naso-pharynx, consisting of infiltration of the middle ear mucous membrane with round cells, overfilling of the blood vessels, desquamation of the epithelium at different places, perforation of the drum, etc.

The same author has increased our knowledge of ear diseases in acute leukemia by a critical review of the whole literature on this subject, as well as by the addition of 5 cases examined microscopically by himself. Although in leukemia, the changes affecting the inner ear are the most striking, the middle ear often shows characteristic, specific changes. Of the fifteen cases including his own which were examined upon an exact anatomic basis, the middle ear twice showed nothing particular, and was once found normal. In the remaining cases, the following pathologic changes were found in the middle ear: free extravasation of red and white blood cells or the remnants of them, either in the middle ear alone or at the same time in the antrum mastoideum; masses of lymphocytes in the vessels of the mucous membrane of the middle ear, sometimes in greater numbers, sometimes in greater extent; infiltration of the mucous membrane of the middle ear with lymphocytes or hemorrhages; in one of his own cases there was found a fibrino-purulent exudate with numerous colonies of bacteria. Schwabach concludes that the presence of diseases of the ear is not caused, as Gradenigo thought, by a previous or simultaneous inflammatory process in the ear, but that the leukemic exudation of lymphocytes

can come about entirely independent of any such processes, while the hemorrhages are to be referred to changes in the blood vessels similar to those caused in other organs.

In regard to the relation of middle ear disease to measles, the works of Bezold[39] and Rudolph[40] show that otitis of measles presents certain characteristics both in an anatomic and in a clinical sense. They regard the purulent inflammation of the middle ear cavity as an integral, specific part of measles, and base their opinion on material consisting of sections of the ears of patients dying of measles. "The entire form of the inflammation of the mucous membrane of the middle ear, its varying extent in different regions, the macular and punctate injections, the extravasation of blood, and perhaps the small granulations rapidly forming in different places lead to a comparison of the mucous membrane diseases to the exanthema of the skin, and the constant pathologic findings of this form are best explained if they are regarded as analogues of the exanthema of the skin."

Lomel[41], in view of the report of 25 postmortems in the clinic at Basel, had to abandon the opinion that an otitis media was a part of the picture of a diphtheritic disease of the respiratory organs; the middle ear inflammation is no genuine disease but a part of the general infection. Among his postmortems, there was only once a normal middle ear, but in the other cases there was every possible quality of otitis media, though only twice specifically diphtheritic. The correctness of Lomel's view was later confirmed by the much more extensive investigations of Levin (l.c.) at the ear clinic at Graz. In 63 per cent. of the diphtheria cases examined, Levin found an involvement of the ear, a frequency which depends chiefly on the youth of the patients. In children under 5 years, he found otitis in 84 per cent of all pos tive cases of diphtheria, in children between 6-10 in 13 per cent. 11-15 in three per cent. and 0 per cent. in those above 15. The nature of the concomitant otitis was usually a simple exudative otitis media, rarely was it a tubo-tympanal catarrh. In a very few of the cases, symptoms of a severe form of otitis acuta appeared. Very seldom was there a spontaneous perforation of the drum. Specific, diphtheritic inflammation of the middle ear was found very rarely by Levin; the

otitis of diphtheria usually presented the pathologic-anatomic picture of the ordinary otitis media. We have already referred to Levin's view that it is of a toxic nature and hematogenetic. Levin's law as to the predisposition to diseases of the ear in children in the first 5 years of life, who are suffering with diphtheria, has lately been confirmed by Stangenberg[42] on the large material of 1000 cases of diphtheria in the Epidemic Hospital at Stockholm.

As to the relation of Bright's disease of the kidney to the accompanying hemorrhages from the ear, we have the pertinent work of Haug[43]; "Since we know that all these hemorrhages occur without any special gross lesion, by diapedesis, we can assume that the same happens in aural hemorrhages. We have, therefore, in the ecchymoses of the drum, in the bloody suffusions of the mucous membrane of the middle ear, in the labyrinthine hemorrhages, and in the hemorrhagic transudation into the middle ear itself, the pathologic-anatomic finding reported in the literature, analogous to the above-mentioned hemorrhages into the organs, on a nephritic basis."

Jehangir S. Cursetji[44] made the first report on the complication of the bubonic plague by an acute otitis media. According to him, the otitis in plague becomes suppurative. In the fibrinous, serous, sero-sanguinous, or sero-purulent aural discharge, he was able to demonstrate the pest bacillus. The aural complication was found in 2 per cent. of the cases treated in the hospital, but in 10-20 per cent. of cases with buboes in the region of the parotid or submaxillary glands or the throat. 70 per cent. of the cases ended fatally by meningitis.

From Schwabach's[45] work on tuberculos s of the middle ear we find that the otitis media tuberculosa may appear under the clinical picture of an acute otitis media, and that there is a relative frequency of paralysis of the facial in tubercular middle-ear suppuration. According to Schwabach's calculation, which is based on a material of 3737 suppurations of the middle ear, paralysis of the facial occurred 18 times as frequently in tuberculous as in non-tuberculous suppurations.

Scheibe describes a form of chronic middle ear tuberculosis

which does not manifest itself like the "phthisical" form in rapid destruction of tissues, but may remain stationary for years. In such cases he found transitory formation of a gray adhesive deposit on the mucous membrane of the vicinity, which microscopically resembled the fibrinous deposit in otitis externa crouposa, but unlike that, was not detached in toto, but gradually disppeared and seldom returned. Numerous tubercle bacilli were found within the deposit. The histologic examination showed that it was a fibrin-like mass, which did not accept Weigert's stain, and which is to be regarded as identical with the fibrinoid of Schmauss and Albrecht (Virchows Archiv. Bd. 144, Suppl-Bd, 1896).

"The matrix is a clear, granular mass, partially fibrinous at the periphery, which stains little or not at all, and which does not accept the Weigert stain. In this granular, globular mass, are found certain large bodies of irregular jagged form which are differentiated from their surroundings by a somewhat stronger susceptibility to stains. The jagged bodies often long are perhaps partially explainable as remnants of cells, but partially, by their arrangement, they resemble connective tissue elements. Vessels are nowhere found in the deposit. Although in one case, distinct cells are completely absent, in another in which the granular matrix was present in smaller amount and more deeply stained, cells with a large, round, pale nucleus were at least indicated, and here and there, scattered over the entire specimen, were found numerous well stained leucocytes with a nucleus or several fragments. At certain places in this case, the tissue structure was plainer, so that it seemed as though there was neurotic tissue at these places."

In the peculiar anatomic relations of the child's mastoid we have an explanation of the fact that the disease of the child's ear has a special place, and also that the ear is more disposed, in general, to disease than is that of the adult; in the same way the width of the child's tube enplains why in tuberculous disease of the lungs and upper air passages infectious material enters the ear so easily and causes a tuberculous disease of the same. That middle ear tuberculosis is probably much more frequent in children than is generally believed, has only recently been shown by

Licci.[47] By injection experiments he has shown that many otitides in children, which progress under the clinical picture of an ordinary chronic suppuration, are of tuberculous nature, and the tuberculous character of the middle ear suppuration is only obscured by the presence of other germs. Of course we may ask whether the simple suppuration was not the primary and the tuberculous the secondary condition. As to how far the tuberculous taint is responsible for the presence of middle-ear disease in children has been investigated thoroughly by Ostmann[48] on the school children of the district of Marburg. He comes to the conclusions that the tuberculous taint favors the origin of ear disease and exerts all unfavorable influence on it in proportion to the severity of the taint. The connection between the tuberculosis of the nearest blood relations and the predisposition of tuberculous tainted children to diseases of the sound conducting apparatus must be sought, according to Ostmann, in the increased irritability of the mucous membrane of the upper air passages, especially of the nose and throat; and the further fact that in them the diseases of the ear invariably lead to severer disturbances in hearing than is generally the case in similar diseases, can be explained only by the supposition that their constitutions possess a lessened resistance to injurious elements.

As to the relation of the middle ear to abdominal typhus, Suckstorff[49] found eight cases of otitis media (7.7 per cent.) in 90 of typhoid during the typhoid epidemic at Rostock in 1900, which can be called a severe one on account of the high percentage of mortality, 13.4 per cent. These began between the 17th and 28th day of the disease, confirming our experience. There was mastoid involvement in only two cases. one of which was complicated by extradural abscess in the posterior cranial fossa and destruction of the sigmoid sinus.

That middle-ear disease is more frequent in typhoid children than adults is shown by the statistics of Schuhmacher[50], who thereby performed a considerable service, since no comparison of the ear diseases of typhoid children, etc., has heretofore been made. Of 100 ears of typhoid children only 29 were normal. In 31 cases the mild character of the

inflammation manifested itself in injection of the vessels of the handle of the hammer. Simple catarrhal inflammation of the middle ear and the Eustachian tube was present in 32 cases, acute middle ear suppuration in 11 cases, of which 6 were bilateral. These middle ear diseases appeared 4 times in the first week, 11 times in the second, 11 times in the third, 14 times in the fourth and 4 times in the other three weeks. A glance at these numbers shows that the frequency of middle-ear diseases in typhoid children is not explainable by the great frequency of middle-ear affection in childhood; it must, perhaps, be admitted that the ear of a child with abdominal typhus is in more danger than the ear of an adult with a similar disease.

CAN PRACTICAL CONCLUSIONS BE DRAWN FROM THE FINDINGS WHICH HAVE BEEN SUPPLIED BY THE LABORS OF THE LAST DECADE IN REGARD TO THE RELATION OF THE DISEASES OF THE MIDDLE EAR TO OTHER DISEASES?

This question, as far as infectious diseases is concerned, can at once be answered in the affirmative. The special feature is the prophylaxis, and in the same sense in which we have already spoken in the discussion of the bacteriology of the diseased ear.

As far as the prognosis of otitis media is concerned we have shown above what an important point in making a correct diagnosis is furnished by the recognition of the bacteria found in the aural discharge. The fact proven by Schwabach that tuberculous middle ear suppuration may resemble an ordinary acute otitis media is an additional proof of the importance of a bacteriologic examination of the discharge in those cases where we wish to avoid gross prognostic and diagnostic errors.

The fact lately insisted on by Bezold and Rudolph, that an otitis media is an integral part of every case of measles, makes it the duty of the general practitioner to pay special attention to the ear in every case of measles, even when absence of aural symptoms makes an examination of the ear seem superfluous.

As to the practical value of the facts concerning the rela-

tion between the ear and diabetes, the statistics of Körner (*Deutsche Arch. f. Klin. Medizin Bd.* LIII., S, 588) showing that acute middle ear suppurations lead to temporal bone disease much more frequently in diabetics than in patients not suffering with this disturbance in metabolism demands the greatest solicitude for the free exit of the pus from the middle ear. If the suppuration does not heal in a short time, but continues in an undiminished amount, considering the great danger of the rapid extension of a destructive bone process in the mastoid, the opening of the latter is indicated even when no exact symptom is present of the existence of a suppurative process in the bone, although Kuhn, Körner and Kirchner have shown rapid necrotic processes can run their course in the mastoids of diabetics, without any external signs of inflammation.

While König and Kraske have successfully fought the position of the surgeon against operations on diabetics and have shown it to be untenable, the change of view brought about by them must itself suffer change, in the prognosis of the aurist as to operations on diabetics, the more so as we have gradually come to recognize in treating mastoid affections of diabetics, that we are not dealing with a specific affection but with a form of disease which shows an increased tendency to malignancy on the basis of diabetic anomalies of metabolism.

How this change has been brought about is shown in the numerous reported results of operations on the mastoids of diabetics which have appeared in the last decade.

The favorable results have been overwhelmingly in cases of mild diabetes, while in severe cases with positive Gerhardt's ferric-chloride reaction, the operation has often caused fatal coma.

That the danger of the coma can be avoided by an antidiabetic diet preceding the operation is generally accepted. The greatness of this danger finds full expression in the increase of excretion of sugar however transitory, after the operation, in spite of constant diet, which has been reported by some authors even in cases of mild diabetes. The danger to the diabetic in the operation seems to consist in the fact that an unstable metabolism is so acutely disturbed by the

operation that an equalization of this disturbance does not always follow in time.

Many valuable observations have been made during the last decade on those diseases of the middle ear in whose origin the mechanical factor of an inequality of the air pressure in the middle ear and that of the atmosphere, or surrounding medium, plays a causal role,

Koch made a valuable study of the influence of diving on ear diseases by examing 29 divers of the diver's course at Wiesbaden. He saw the following pathologic changes:

1. Retraction of the drum, which was greatest at the membrana Schrapnelli, even causing perforation at that position.

2. Vasomotor disturbances of different degrees, manifesting themselves in the different forms of more or less prominent congestion of the drum, and sero-sanguinous transudate into the middle ear, with formation of red vesicles behind the drum.

3. Rupture of the drum (only in atrophic drums).

4. Acute middle-ear inflammation caused by the high pressure in the nasopharynx driving the mucus situated in or before the mouth of the tube into the middle ear.

Alt, Heller, Mager, v. Schröter[52] have studied the pathologic action of change of pressure on the ear and especially the middle ear by examining caisson workers and by experiments on animals. The etiologic factors for the pathologic changes are on the one hand the mechanical factor of the unequalized difference in pressure in the middle ear and the surrounding space, and on the other the gas emboli appearing in the bloodvessels after sudden decompression. The most important for the disturbances in the region of the middle ear is of course the first factor. During the compression, which causes a negative pressure in the cavum tympani, by preventing the movements of the tube, we may have retraction of the drum with livid discoloration, ecchymoses, ruptures and hemorrhages into the middle ear. Likewise, in decompression, as a result of the failure of equalizing pressure, we have bulging of the drum with injection, hemorrhage into the drum and meatus and rupture.

The mechanical factor of the inequality of the intratympanal and external air pressure plays often a considerable

role in the origin of the middle ear diseases caused by adenoids. In addition to the gradual absorption of the air in the middle ear, the following factor, according to Barth, comes into consideration as the cause of the great retraction of the drum: during every act of swallowing the soft palate is lifted in a high bow, in many cases reaching higher than 1/2 the height of the choanae. During this muscular act, the adenoids are pressed in all directions, and of course toward the openings of the tubes so that they are completely shut off from the entrance of the air during the act of swallowing. In addition to this, there is the sucking action exerted by the soft palate as it returns to its postion of rest. This is caused by the sinking of summit of the palate before it is separated from the posterior pharangeal wall.

The changes on the drum and the middle ear caused by mechanical factors, belong to those caused by the action of explosions. Muller in about one half of a large number of cases examined immediately after artillery practice, found hyperemia and even hemorrhage into the drum, and in one ear, previously normal, found a vesicle of blood.

IN REGARD TO THE MIDDLE EAR INFLAMMATIONS WHICH HAVE BEEN ASCRIBED TO CERTAIN NERVOUS INFLUENCES, there has always been a certain scepticism, the more so as the experiments to produce pathologic changes in the middle ear after section of the trigeminus, after analogy of experimentally produced eye diseases, have led to contradictory results, (Hagen, Gellé, Berthold, Baratoux). The case of Asher[55] is of importance, since that author does not use the mystical "trophic" nerve fibres in explaining a severe exudative otitis media that occurred 3 weeks after resection of the 2nd and 3rd branches of the trigeminus (on account of neuralgia).

According to him, the otitis media came about in the following manner: vasomotor disturbances caused either by the destruction of the vasomotor fibres running in the trigeminus or those which rising in the sympatheticus, later join the trigeminus, or by reflex excitation of the vasodilator, caused by processes at the stump of the trigeminus. Then entrance of germs into the thus sensitive space from the nasopahrynx, diseased in this case, through the permeable tube. In this

case, hypothesis has a wide play, and it is possible, in view of the existence of a purulent inflammation in the naso-pharynx, that the otitis media was a complication independent of the resection of the trigeminus.

Kraus[56] in considering the otitis media following resection of the trigeminus, agrees with the view of Asher that the tissue possessed a weakened resistance on account of the loss of nervous influence, and thus became a favorable medium for the growth of germs, without giving any satisfactory reason for his opinion.

The general pathology of the ear has received an incalculable aid both theoretically and practically, by Braunstein's and Buhe's[57] proof of the non-existence of vessel communication between middle ear and labyrinth, although this had been generally accepted since the comprehensive investigation of Politzer. Apparently everybody had accepted Politzer's view, although Siebenmann as early as 1890, in his corrosion experiments on the temporal bone, could not convince himself of this communication, and Eichler in 1892, in his beautiful investigation on the blood current in the human labyrinth, had directly contradicted Politzer's statement. The microscopic preparations of Braunstein and Buhe—stained with eosin and hemotoxylin, show that the blood vessels passing from the mucous membrane of the middle ear into the bone run to the inner layer of the compact substance, and end here or bend upon themselves and run parallel to the bony lamellae. They never penetrate the cochlear capsule or make anastomoses with the labyrinthine vessels.

SPECIAL.

ANOMALIES OF DEVELOPMENT AND MALFORMATION, whether they affect only special parts of the middle ear or have led to a complete change of form of the same, with or without involvement of the outer ear, are not uncommon; nevertheless the individual anatomic findings should be reported because, on account of lack of operative treatment of these abnormalities, we very seldom have an opportunity of completely studying the abnormal anatomic conditions in each case. Of

the anomalies affecting the contents of the middle ear, those of the ossicles are of special interest.

So, e. g., Tomka[58] found in one case that the stapes had only one crus; at the same time the eminentia pyramidalis and the musculus stapedius were absent. In a second case, the stapes had the form of a triangular piece of bone. It was a persistence of the original form of a bone, insofar as the space formed by the passage of the art. perforans staped. between the cura of the stapes had not developed. Furthermore, he described[59] an interesting case of supernumerary ossicles. In one case there was, between the anvil and the medial wall of the middle ear, a piece of bone 5mm. long covered with mucous membrane, and having a smooth surface. The origin of this structure is to be referred to the development in the inferior maxillary process, in addition to the cartilaginous anlage for the hammer and anvil, of a third cartilaginous anlage in the immediate vicinity of the anvil, from which the accessory bone was developed. In Hammerschlag's case[60] of a 20 year old woman with middle ear diminished in every dimension, the hammer and anvil were of normal size while the stapes had a small foot plate, corresponding to which, the oval window was smaller than normal. Furthermore the crura of the stapes were so pressed together that they gave the impression of a single crus. In the left ear of a syncepha us janiceps asymetros child was found a persistent arteria stapedia, a vessel which, after its appearance in an earlier epoch of embryonal development, soon disappears. In this case, it arose from the arteria carotis interna and piercing the floor of the middle ear, entered the latter. From here, enclosed in a bony canal, it passed in front of the promontory, through the stapes toward the roof of the tympanic cavity where it passed through an opening in front of the hiatus spurius into the cranial cavity. Here it ended in a lateral branch that anastomosed with arteria meningea of this side, and in a medial one that anastomosed with the vessels of the orbit and branched on the cerebral surface of the large pterygoid processes of the sphenoid. A hitherto undescribed anomaly of the stapes, viz: a congenital adhesion of its posterior bar to the wall of the Fallopian canal, has

been described by Politzer[62]. It was in the temporal bone of a deaf-mute. The niche of the oval window was very shallow, and thereby the posterior bar was brought closer to the facial canal than normally. At this place, there was a dehiscence in the wall of the facial canal, and the posterior crus of the stapes was directly adherent to the wall of the canal.

The cases of D. Kaufmann[63] and Haug[64] dealt with anomalies that concerned the entire middle ear.

In Kaufmann's case the specimen came from an 85 year old day-laboring woman, accidentally killed—the malformations were confined to the external and middle ears. The conchae of both sides were badly distorted, the external meatus was absent, the os tympanicum on both sides was completely malformed, i. e., in its place was a tumulous mass of bone, which passed over into the surrounding bony part (root of the processus zygomaticus and mastoid). On neither side was there a trace of a drum. The tympanic cavities were abnormal in that the Eustachian tubes did not extend so far posteriorly. In the tympanic cavity were a number of irregular spaces bounded by plates of bone and connective tissue, which were joined to the ostium tympanicum tubae and the internal wall of the middle ear, in the angle between the anterior and external semicircular canals. With the exception of the absence of the antrum, the mastoids were normal. While in the right ear the hammer and anvil had developed as a single piece of bone, the ossicles of the left, bound together by normal articulations, formed a body which lay in the recessus epitympanicus—enlarged in every direction—while the ossicles did not seem to lie in the tympanic cavity itself, since the handle of the hammer was not developed, and the long process of the anvil was malformed." "The processus longus of the anvil was curved forward and inward, and was thicker than normal and lengthed. An articular facet for the stapes was not visible."

Of importance was the fact that the carotid canal did not extend as far posteriorly as normal, so that the distance between the knee of the carotid canal and the anterior margin of the fenestra ovalis was less than is usually the case. The

whole labyrinth as well as the nervus acusticus was found normal after a careful microscopic examination

In addition to this case, Kaufman described another specimen from the pathologic-anatomic institute at Vienna, in which there was likewise an absence of the external meatus, drum and os tympanicum; this specimen showed a higher degree of malformation in so far that in the middle ear there was only a remnant of the tube to be found, while all the rest was a compact bone, containing little spongiosa.

In both cases, Kaufmann considers that the disturbance in development took place in a very early time of embryonal life, viz., at a time when the external meatus had not developed. While in the first case, "later—from the stage of the absent meatus—the ear had as a whole developed normally" in the second case, on the right side, the development of the ossicles had entirely failed.

While Kaufman, in regard to the cause, seems to concur with the view of Virchow that in these malformations, it was not a question of a simple retarding of development, but rather that an irritative process unknown to us for the present must be assumed, it seems to us that his first case with its multiple bony and connective tissue plates in the middle ear which can be regarded as the remnants of an intra uterine inflammation speaks for the correctness of that assumption.

In the case reported by Haug, (l. c.,) of a unilateral congenital malformation, the specimen came from a female child that died shortly after birth. Accompanying the normal formation of the labyrinth, there was a high degree of malformation of the concha, a stenosis of the external meatus nearly reaching an atresia, a small middle ear with a badly formed drum; all ossicles were abnormally developed, the hammer was inserted into the drum, and articulated with the formless anvil. The stapes was firm in the oval window, but was not articulated with the anvil. The tube was represented by a solid piece of bone, the musculus stapedius was absent, the facialis was very thin. Furthermore, the pars squamosa of the temporal bone had never developed, the lower jaw on that side was malformed, and this was the cause of the palpable asymmetry of the face."

As to the DISEASES OF THE DRUM, a new light has been thrown on the etiology of the epithelial pearls of the drum first described by Urbantschitsch by the discovery of such pearls in a linear paracentesis cicatrix by Habermann (v. Schwartze, Handbuch der Ohrenheilk. Bd. I. S. 238) and Grunert[65]. The depicting of such multiple pearls by Urbantschitsch makes it appear without doubt that those structures are true, perhaps congenital, tumors. This view is strenghtened by the complete microscopical findings of Wendt, etc. The case of Habermann and of mine, on the contrary, shows that these structures have no special etiology. If especially in our case, in which the new formations did not appear until after the cicatricizing of the paracentesis wound, we would think of a causal relationship between them and the paracentesis incision, the results of the experimental and clinical investigations of Kaufmann[66] and of Blumberg[67] would give a certain support to the view, that these structures were derived from epidermis which was accidentally implanted into the tissues of the drum by the paracentesis section. But the epidermis which in the socalled mammiliform perforation frequently grows over from the free surface into the opening (Katz, etc.) must also be thought of as a possible origin of these structures. In this way also, its position is shown as a structure differing from cholesteatoma not in any principle but in degree, although Leutert has given us a very plausible explanation for the origin of a distinct form of cholesteatoma in this way, viz., by transplantation of pieces of epidermis of the drum into the granulation tissue lying behind it, by means of instrumentation. To the new formations of the drum also belongs a case of osteophyte formation on the handle of the hammer, which Singer[68] reported. The drum showed the objective signs of chronic inflammation; at the location of the hammer was a semicircular, white, projecting bow, with very small jagged projections, which seemed to include the auterior fold and the short process, and reached to the middle of the periphery of the drum. Palpation with the probe showed that it was a case of osteophytes. In our opinion, it was a case of inflammatory new formation. We consider the osteophytes as the products of

an ossifying inflammation of the soft covering of the handle of the hammer, after the analogy of osteophytes of the meatus, which in chronic otitis media often appear as sequelae of a concomitant ossifying periostitis of the meatus. We do not think that Singer's case was one of congenital exostosis of the hammer for this does not usually reach the size described here, but appears as small needleformed and spinous projections of bone.

Out of the profuse discussion of the last decade concerning trauma of the drum, we select, as belonging to the very rarest occurrences, a case of emphysema of the membrane, which Haug (1. c.,) has described. The emphysema, in this case a part of an emphysema of the regio mastoidea, was characterised by several vesicular prominences of the cutis layer from the size of a lentil to that of the head of a pin, which in part were bloody. The mucosa and substantia propria of the very delicate drum were ruptured by the sudden entrance of air into the middle ear.

Corradi[70] has made a very complete study of ruptures of the drum by indirect force, and came to the following results. "While the perforations of the drum by indirect causes, as the result of air pressure in the external meatus, are usually located more or less toward the middle, near the lower end of the handle of the hammer, those resulting from blows on the head or fracture of the bone are often located near the border, so that there seems to be an actual detachment of the drum from the bony part." Pritchard[70b] has reported a case of fracture of the handle of the hammer by indirect force.

The literature of the last decade has been extraordinarily rich in extravasations into the drum. Among them should be mentioned a new form of multiple ringformed (the ring is not closed) hemorrhages into the drum, which Körner[71] has described. "On the grey, partially grey-red, background, these brownish red rings give a picture that easily reminds one of a panther's skin." It was a case of influenza otitis. The presence of extravasations into the drum and meatus has been reported by Tomka[72] in a case of acute otitis media accompaning the morbus maculosus Werlhofi, and by Sugar[73]

in a case of recurrent purpura hemorrhagica; the latter considered these extravagations purpura eruptions.

The heretofore unobserved presence of true varices on the drum has been demonstrated by Rohrer[74]: There developed in a 7 year old boy, alternately on the right and left sides, in the membrana Schrapnelli and the superior posterior quadrant of the drum, numerous varices of the size of a grain of pepper and a small pea, which spontaneously disappeared, leaving a scar.

As to the primary inflammatory processes of the drum the repetition of a case, reported by me[75] from the Halle clinic, of interlamellary abscess of the drum seems to be justified, because in this case a ray of light has fallen on the ordinarily dark etiology of the abscess. The finding on the drum was as follows: Injection of the vessels of the manubrium mallei. A group of vessels ran from the processus brevis directly upward, and another upwards and backward. In the region of the umbo, were two prominences of the size of the head of a pin, which were yellow, tipped with a light reflex. A third prominence, smaller and not shining, lay somewhat higher, and in front of the handle of the hammer. On opening the small protuberances with the paracentesis needle, a drop of pus came from each, whose bacteriologic examination showed the presence of numerous streptococci.

The traumatic origin of the interlamellary abscesses was first suggested by Schwartze (Lehr. d. chir. Krankh. d. Ohres 1885 S. 119 and 120); he reported a case of interlamellary abscess which followed the application to the drum of a dilated solution of tincture of iodin, and a second case of abscess whose origin he refers to the application to the drum of a solution of 10 grains of silver nitrate.

In our case also, in all probability a trauma was the cause. The patient, while trying to free his ear from wax, injured his drum with a needle-like instrument; the lesion was probably the port of entrance of the streptococcal infection into the tissues of the drum.

Katz[76] has made us acquainted with the formation of spaces or cysts in the stria malleolaris. In longitudinal sections of the drum, he saw, with the lens magnification,

a detachment of the cuticular layer from the subjacent propria, along the entire handle of the hammer. In this space large masses of leucocytes or fibrinous extravasations were found. This space formation, to which the large amount of subcutaneous tissue in this region predisposes, is considered by Katz to be the cause of the stria malleolaris becoming indistinct so rapidly in inflammatory processes on the drum.

That the persistence of perforations in the drum after the cessation of middle ear discharges is caused by the epidermis of the meatus growing over the border of the perforation, is shown by microscopic preparations which Politzer[77] demonstrated before the Third Austrian Otologic Congress.

Let us turn to the important findings concerning the pathologic anatomy of the middle ear that the last decade has produced.

A valuable work by Barnick[78] gives us a good view, especially of the microscopical details, of injuries of the middle ear in consequence of fractures of the temporal bone. These usually consist of hemorrhage, fracture of one or more walls, rupture of the drum, etc.

Stillkraut has given us a dissertation on the presence of hemorrhage in chronic nephritis.[79] He thinks that they are caused by disturbances in the nutrition of the vessel's walls, from which results a great permeability of the same.

We will mention only a few of the most noteworthy cases from the very rich literature on foreign bodies in the middle ear.

1. A case of Camerer[80], in which a blade of straw had passed through the Eustachian tube into the middle ear.

2. A case of Schmiegelow[81] in which a stone covered with earth was pressed into the middle ear by unskilfull efforts at extraction, resulting in a tetanus infection.

3. A case of Ostmann[82], in which a piece broken off from the point of a knife remained 12 years in the mastoid without any symptoms and had caused aural discharge only for a year.

4. A case of G. Trautmann[83], in which a cherry seed had been pressed into the pharyngeal end of the right tube.

5. A case of Ponthiere[84], in which the foreign body, a bent

piece of copper wire, 12 mm. long, had lain in the middle ear 16 years and had caused suppuration with polypus formation.

6. A case of Richter[85], in which a white piece of bone had lain 24 years in the ear, and had entered it by a malposition of a bony defect running from the external meatus into the antrum mastoideum.

7, A case of Haug[86], in wh ch a plug of cotton was pushed too deep into a radically operated ear, and by means of a forceps or hook was thrust through a thin place on the floor of the middle ear along the tissue between the tuba Eustachii and the musculus levator palati mollis as far as the peritonsillar issue, where it came to a halt, and led to an abscess formation, from which the foreign body was extracted by an incision.

8. A case of Schade[87], in which the wandering of the foreign body was reversed, viz., from the mouth through the middle ear outward. The patient was a 19-year-old merchant, who had drawn out a bent nail from a cigar box with his teeth and accidentally swallowed it. It was not possible to extract the foreign body, and for eight days he suffered rather severely with his throat. Four days later, following a forcible movement, he experienced a severe pain in the left ear caused by a severe otitis media. Three days after the beginning of the pain in the ear, it was possible to remove the nail from the ear. It was 12 mm. long and 1 mm. thick, and bent at a right angle in the middle.

As to the acute otitis media, a case of Haug[88] shows that the exudate vesicles in acute catarrh are not always caused by a bulging of certain parts of the drum. In this case of influenza otitis upon which an autopsy was made, a serous exudate in the superior posterior part of the middle ear had emptied itself through the posterior fold, which was shut off by fresh membranous adhesion, into the drum, and after tearing the mucosa and substantia propria, had bulged the cutis out in the form of a large vesicle.

As to the pathologic anatomy of otitis media acuta suppurativa, our knowledge has been considerably increased in regard to the teat-like projections of the drum so often seen in this disease, especially in the posterior half, on whose summit frequently small granulations are observed. Bezold

explains the e granulations as mucous membrane pressing out around the border of the perforation, which, according to Grünwald, is often covered with the mucous epithelium of the middle ear, or with the epidermis of the drum. Haug says they arise by hernia like prolapse of the mucosa through the small opening of the perforation, Katz[89], on the basis of his microscopic investigations, comes to conclusions differing considerably from the above authors. Whether this theory is good in all cases, or whether a variety of factors caused the formation of the bodies may be left unsettled. At any rate, Katz has given us a clear, positive anatomic finding, which is of more value than all speculative discussion, and therefore deserves to be reported in full. "If one examines microscropically a cross-section of such a tympanic prominence several interesting points will be observed. First, it is evident that it is a densely inflated granulation traversed by a narrow canal, and is covered with a greatly inflamed epidermis or rete, the whole taking its origin from the mucosa of the middle ear or the drum. Furthermore, the canal that lies within it is formed or lined by a layer of epithelium which is similar to that covering the granulation. This circumstance is explained by the examination of serial sections, which show that the tympanic epidermis has grown into the perforation opening. Furthermore, in addition to the epithelial-lined canal there is found within the tumor an excavation caused by the high pressure of the exudate. Finally, the upper layers of the epithelial covering of the tumor, which is in continuity with that of the external surface of the drum, show a considerable cornification, and between the lamellae at many places there are very plain pustules (nests of leucocytes). As to the etiologic factors of this cone-like body, the following are suggested by Katz:

1. A very narrow, high perforation.
2. A constant exudate in the middle ear.
3. Resistant tympanic epidermis.
4. Inversion of the epidermal border into the narrow perforation, making it still smaller.

The influence of these etiologic factors is very plausibly explained as follows: "In consequence of the irritation that the difficult passage of the thick pus exerts on the neighbor-

ing tympanic mucosa, there is set up a circumscribed granulation formation. This, in its further development, causes the adjacent substantia propria to disintegrate or become necrotic, but finds in the perforated but still resistant rete and epidermal layer an obstruction of such a character that the only possibility is for them to be pressed forwards like a hernia, and to grow with it. The small perforation can of course not be widened sufficiently, especially since the opening has been malplaced by the inversion of the epidermis. This inward misplacing of the opening easily explains the ability and the tendency of the leucocytes to press into the horny layer by way of the lymph channels in the reticular layer and the formation of pustules." Although Bezold[90] has lately agreed with Katz in regard to the anatomic explanation of that structure—above all in regard to the epithelial lining of the canal in the cone—he regards Katz's statement that the growths which are bare and those which are covered with epithelium are different kinds of structures, as superfluous and leading to error. We cannot agree with Bezold's view. We consider that the best way to obtain greater clearness in regard to the interesting tympanic cones is to get exact anatomic date concerning the individual kinds of those structures. From the variable results of the investigations of earlier authors, it seems probable that those bodies are not to be considered as unvarying in pathogenesis and structure.

I have already indicated the possibility that by the anatomic processes just mentioned, the ground is laid for the subsequent formation of the epithelial pearls in the drum. Whether these processes, when the acute suppuration does not heal, can also give rise to formation of cholesteatomata, and whether the origin of cholesteatomata in many cases of more medially lying perforations is to be ascribed to the same reason, can be shown only by further investigation.

The practical application of the above anatomic facts suggests itself.

First, as a prophylatic measure, we must prevent that factor, which with thick pus, predisposes to the formation of the cones, viz: small high perforations. We will accomplish this by dilatation of the narrow opening with the paracentesis

knife, or with the galvanocautery. If such a tympanic cone has already formed, the danger of retention of pus constrains us to remove it. This can be accomplished with caustics and the cold snare only when we are dealing with bare granulation tissues, and not with that covered with epidermis. In the latter form, where there is a fistulous canal, lined with epidermis (Katz), these measures are often of no avail; in such cases good is accomplished by the galvanocaustic destruct on of the cone.

The following may be mentioned as of peculiar individuality, from the anatomic investigation of the acute suppuration of the middle ear, during the last decade.

Katz[91]: A case of hypertrophy of the mucous membrane of the middle ear, with cone like growths of the mucous membrane around the round window, which had drawn the membrane outward.

R. Panse[92]: Path from the middle ear to the internal ear. Discharge of the pus through the annular ligament of the stapes above and below, partial destruction of the membrane of the round window, collection of pus at the base of the scala tympani; reaching in places to the bony spiral lamella. Thickening and infiltration of the wall of the utriculus with round cells; filling of the perilymphatic spaces of the semicircular canals with fibrin and round cells; both aqueducti contained pus, also. Membrana Reisneri destroyed, filling of the parts of the scalae lying near the modiolus with pus; the spiral ganglion canal permeated with round cells; the acusticus entirely suppurated, the facialis permeated with round cells but preserved.

Scheibe[93]. Progressive disease of the labyrinthine capsule consisting of necrosis, rarefying osteitis and softening of the bone. The perforation into the labyrinthine cavity took place at three places, at the posterior and the superior semicircular canals and at the superior and inferior borders of the stapedial annular ligiment. The perforation at the semicircular canal was caused by the rarefying osteitis, and at the oval window by necrosis.

As to the involvement of the mastoid in acute otitis media, I have already indicated how much our views have changed as to whether the mastoiditis accompanying the

acute otitis of diabetes is primary or secondary. The characterization of the diabetes mastoiditis as primary has been almost universally abandoned. The same is true of the mastoiditis of influenza, in regard to which in 1894, Lemcke[94], Jansen and Körner (ibid) stated that the disease was especially located in the mastoid. The latter claimed that the disease was chiefly located where there was diploe, especially at the posterior border of the tip, and in the vicinity of the sinus transversus. Although Eulenstein (ibid) in regard to the view of Lemcke, makes use of the clinical fact that frequently after removal, by an operation, of the bony foci in the mastoid, the suppuration in the middle ear at once ceased, I can urge against this use the fact that the same experience is met in many cases where there is no doubt about the secondary nature of the disease of the bone. That Brieger's claim that a case observed by him was a primary disease, viz., an osteomyelitis of the mastoid, will not stand a searching criticism, I have already shown. Against his view, is the presence, however transitory, of a disease of the middle ear, which leads to the belief that it was a case of pneumococcus otitis. Unfortunately, the lack of a bacteriologic examination prevents an explanation of this case that is without objection.

So far no other author has had a case similar to Hessler's[95] case of "rarefying ostitis" of the mastoid, which followed an "otitis externa ex infectione", and which was microscopically characterized by brownish discoloration of the spongiosa, made brittle by decalification of the bone, and by complete absence of inflammatory changes on the mucous membrane of the mastoid cells.

Although Körner[96] wished to divide sharply the bone diseases complicating acute otitis media into "absorbing processes" and necrosis, it must be held against his view that there are frequently transitions between these two forms of the bone disease, and that frequently both processes go hand in hand.

As to the pathologic anatomy of the chronic middle ear suppuration, our knowledge during the past decade has been greatly increased in regard to a large number of points.

There has been a thorough investigation of the paths by

which purulent inflammation of the middle ear passes over to the labyrinth. Among these work, as of first merit, must be mentioned that of R. Panse[97] , the worth of which rests upon the exact microscopic examinations. Concerning the rarity of its presence, must be noted a case of Katz[98] who examined microscopically the path of the inflammation from the middle ear to the labyrinth. The mucous membrane of the promontory of this case was found necrotic at a circumscribed place. The bone subjacent thereto was destroyed by caries as far as to the endosteum of the cochlea. In the cochlea was a rich fibrinous exudate. In the case of Manasse's[99] the plate of the stapes furnished the path of the inflammation from the middle to the internal ear; granulations had grown through it, and the cochlea and the vestibular apparatus were filled with granulations and connective tissues, the auditory nerve was infiltrated with round cells, and its fibres separated by exudate.

Of the cases of chronic middle ear suppuration with interesting pathologic findings, we must mention those of Politzer[100], and Zeroni[101]. Politzer studied thoroughly the histologic changes in the mucous membrane in the chronic middle ear suppuration of childhood. He found chiefly papillary hypertrophy of the membrane, consisting entirely of round cells. The epithelium was cylindrical, ciliated, and much higher than the normal epithelium of the middle ear, and the latter was sometimes entirely filled out by the hypertrophied mucous membrane. In the neighboring bony spaces, Politzer found granulation tissue rich in blood vessels, and dilated medullary spaces in the hammer and anvil with similar contents. A direct connection of the latter with the diseased mucous membrane was not always demonstrable. Cystic structures lined with cylindrical epithelium and filled with epithelial cells, leucocytes, and detritus were found by Zeroni in the depths of the soft parts covering the labyrinthine walls of a specimen removed during a radical operation on a patient in Schwartze's clinic. He denied that they were glandular structures, and explained their origin by supposing that, by the hypertrophying of certain parts, certain normal remnants of the original epithelium had been pressed into the depths and there had developed into cysts.

As to the complication of the chronic suppuration of the middle ear, especially the involvement of the ossicles, the works of Grunert[102], J. Orne Green[103], Körner[104], Kretschmann[105], and Pritchard[106], have all confirmed what was previously reported by Schwartze and his pupils, (especially Ludewig), above all, the great frequency of the involvement of both outer ossicles in the general caries of the middle ear, furthermore the preponderance of the caries of the anvil over that of the hammer, and finally the correctness of the former assumption of the localization of the bone diseases on the individual parts of the ossicles. Although we have for a long time known clinically, through certain otoscopic findings after the middle ear suppuration, *e. g.*, cicatrices in the superior posterior part of the drum, through which the head of the stapes could be plainly seen, that caries of the ossicles can heal spontaneously, Kretschmann has given us the anatomical proof of true cicatrication of carious defects in five case, each time on the anvil.

A work of Brieger and Görke[107] gave us new conclusions in regard to the pathologic histology of the ossicles, which are so frequently diseased in the chronic middle ear suppuration.

Although we have heretofore spoken of caries of the ossicles, Brieger and Görke make the sharpest differentation between necrosis and caries. They especially mention the necrosis of inflammatory origin, which frequently accompanies extreme suppuration of the bony medulla. The sequestra are then usually only of microscopic size; rarely does it come to the formation of typical cloacæ and sequestra, as in osteomyelitis.

The inflammation is said to progress from the periosteum to the medullary spaces by way of the Haversian canals. They object to the view of Donalies[107], who considers that the chief etiologic factor of the bone disease is a disturbance in the access of blood to the bone, by compression of the blood vessels by means of the lymph which has been extravasated into the perviascular lymph spaces. In addition to the possibility of the medullary involvement by way of the Haversian canals, an ulcer of the bone starting in the periosteum may work its way downward and directly open

a medullary space. The periosteal foci, even when they involve the entire thickness of the compacta, may heal by cicatrization. Of importance, furthermore, is the proof that inflammatory processes in the middle ear may attack the medullary spaces of the ossicles, directly, with involvement or appreciable alteration of the perisoteum. We may thus have a central, medullary abscess, around which can form a considerable osteosclerotic thickening. Interesting, furthermore, is the statement in regard to the relative frequency of disease of the articular surfaces in middle ear tuberculosis; all stages were observed, exudate into the joint, deposit of fibrin on the cartilaginous surfaces, and even destruction of the bone. The joint was usually affected secondarily by neighboring foci, which spread along the surface. In only one case was a primary, isolated focus found and then it was on the articular surface of the anvil. The origin of hammer-anvil ankylosis from periarticular processes (Kretschmann) seems doubtful to the author. Furthermore, the author shows that hyperostotic thickenings of the ossicles are found without any trace of previous suppuration.

Of importance is a case reported by Katz[108] of rarefying ostitis of both external ossicula, in a one year old child, suffering with chronic otorrhea; the inflamed bone of these two ossicles, both of which showed normal contour, was traversed by numerous, large medullary spaces, in which were found a very cellular, young connective tissue, and numerous dilated blood vessels. According to Katz, the transition to a rarefying ostitis is very easy under predisposing conditions (tuberculosis, scrophulosis, syphilis, etc.), indeed, according to the author, the difference is only quantitative. This form of rarefying ostitis can, on the one side, lead to a disappearance of the bone, and on the other, to osteoplastic deposits (hyperostoses, exostoses, etc.).

An accidental finding by Hammerschlag[109] of a synostosis of the hammer-anvil articulation in the temporal bone of a 35-year old man is to be considered as the residuum of an isolated disease of the hammer-anvil articulation, since there was an absence of other symptoms pointing to a previous disease of the middle ear. The rarity of this histologic

finding justifies its repor . The microscopic examination showed that the hammer-anvil articulation had disappeared at one place. At this place, the ossicles were bound together by bridges of bone, which were of different breadth in different sections of the series, and which in places showed Haversian canals. Otherwise, the articular cavity at certain places, as well as a great part of the articular cartilage, was well retained. The mucous membrane of the middle ear was everywhere intact.

The anatomic proofs of the spontaneous curability of carious ossicles has given us views concerning the treatment which threatened to make the therapy by removal of carious ossicle valueless. It was thought that the spontaneous curability gave a right to adopt a more "conservative" therapy, and it was forgotten that the anatomically proven possibility of healing did not make a healing probable.

If one desires to adopt an exclusively palliative treatment, on account of the spontaneous power of healing, this can be done in the case of children and in cases of circumscribed caries of the long process of the anvil. Here a spontaneous cure is seen not so very seldom. But such a dangerous experiment must never be tried on adults or in fistulae above the short process, which indicates a caries of the head of the hammer, and suppuration in the attic.

We must never forget that, independent of the danger of the attic suppuration, the carious ossicles may form bands upon which the tympanic epidermis can grow into the middle ear, and thus the ground-work for a cholesteatoma can be laid.

To allow both external ossicles to remain in a radical operation, ostensibly in the interest of the auditory function—it has never been shown that in this way better functional results were obtained—has on closer study been shown to be superfluous and even a bluff, at times.

The past decade has given us some new conclusions as well as interesting facts concerning the pathologic anatomy of aural polypi.

Manasse[110] has given the proof of the presence of multinuclear giant cells in ear polypi (as many as 70 and more

nuclei). They are mostly ring shaped, arranged around a focus of degenerated squamous epithelium, into which they send processes. Individual ones contain masses of epidermis or cholesterin tablets. Manasse explained them as foreign body giant cells, produced by the irritation of dead masses of epithelium or cholesterin crystals. A special type of arrangement of the nuclei is not recognizable. Manasse gives two possibilities for the entrance of masses of epidermis into the polyp; first that they "were detached by the inflammatory process going on in the connective tissue, surrounded by granulation tissue, cut off, and so killed;" and second, that the dead mass of epithelium, lying on the surface of the polyp was enclosed during the growth of these. The giant cells arise as well from one cell, as from the confluence of several. The cells of the granulation tissue give the material for their origin, but in one case, in which both giant cells and dead masses of epidermis lay in preformed spaces, it was given by the endothelium of the lymph vessels. The appearance of scales of epidermis in the lymph spaces, is explained by Manasse on the assumption that "comparatively wide lymph spaces end with open lumen on the surface of the polypi."

The same author has made us acquainted with mucous cysts in polypi, containing giant cells[111].

The cysts in the large polypi, lined with different kinds of epithelium, had arisen from involution of epithelium, by parts of the latter being cut off by the growth of the surrounding connective tissue. The epithelium continued to secrete mucus, so that the wall was stretched to a roundish shape. The giant cells found by Manasse in the cysts, sometimes showed a ound form, sometimes were found with protoplasmic processes. Manasse did not regard the giant cells as products of the cystic epithelium, but derived their origin from leucocytes, which he found in large numbers within the cyst.

To the frequently found, so-called cholesteatomata in the aural polypi, Zeroni[112] has added another finding, of value on account of the histologic examina ion of serial sections. He found the epithelium of the surface enormously hypertrophied, in such a way, that large irregular masses of it

grew downward toward the center. These masses, in which numerous mitoses were found, became cornefied only in places, and so formed the structures mentioned. Other processes which can be seen on the same specimens, are interesting, and consist in the structures apparently disintegrating under the influence of giant cells. Zeroni considers the excessive hypertrophy of the squamous epithelium to be a process caused by inflammation, and thinks he is able to explain thereby, in many cases, the so-called primary cholesteatomata. Kümmel[113] does not consider such globular "cholesteatomata" to be cholesteatomata, but explains them as desquamated, visible foreign bodies of epithelial scales taken up by granulation tissue. The explanation of Kümmel cannot be used in the case of Zeroni's bodies, since the latter is an active growth of the squamous epithelium downward.

In a specimen demonstrated by Brieger[113], of a polyp that had grown out through the membrana Schrapnelli, there were found hairs, and around these, and partially incorporating them, were giant cells. Probably, the hairs, during an attic suppuration had entered through a high fistula into the recessus epitympanicus, and there had given the irritation for the formation of the granulations and the giant cells, in this case foreign body giant cells.

We must thank Görke[114] and Brühl[115] for extensive, systematic investigations on aural polypi.

Görke, on the basis of his investigation, considers the division of aural polypi into granulation tumors and mucous polypi to be the only histogenetically correct one. From these two fundamental forms, are derived all others, however much they differ histologically. He comes to the following conclusions:

1. The new formations of the sound conducting apparatus which are classed as "aural polypi" are of inflammatory origin.

2. According to their genesis, they are either granulation tumor or mucous polypi.

3. Consequently, in their structure, they show the histologic peculiarities of granulation tissue, or of the inflammatory-hyperplastic mucous membrane of the middle ear.

4. Processes of new formation of tissues are found in them.

5. In them appear the multiple products of regressive metamorphosis.

6. Of the latter, as peculiar to aural polypi and as of practical importance must be mentioned the "central cholesteatoma formation."

The work of Brühl reports on the use of the new methods of staining of 60 polypi.

Brühl insists that it is not the epithelial covering that characterises the polyp, but the ground substance, and divides aural polypi, according to their structure, into polypus granulation and fibroma.

Of interest is the comparative examination of cells which the author undertook upon aural polypi. Unna's plasma cells, which are often found in the polypus granulations, he considers to be large mononuclear leucocytes, the bodies of Russell, often found in polypi, he regards as derivatives of the large mononuclear leucocytes.

As to the other complications of the chronic suppuration of the middle ear, Bezold's belief that we must regard the hyperostosis of the temporal bone to be a protective wall against the propagation of the suppuration into the cavum cranii, has continually lost ground, and the view of Schwartze, which was abandoned years ago, that the hyperostosis favors the involvement of the cranial cavity by the suppuration, has been generally recognized as correct.

In this way Haug[116] expresses himself, and claims that as a result of the bony opposition to the periphery of the temporal bone with a simultaneous central absorption, i. e., an external condensing and internal rarefying ostitis, the condition for the propagation of the suppuration into the cranial cavity are much more favorable, in consequence of the pus being prevented from pointing outward by the the osteosclerosis. Lemcke (Berl. klin. Wochensch, 1893, No. 37) says the same. The correctness of this view is attested by the statistics of Okuneff[117], who found osteosclerosis in more than half of his case that died of chronic middle ear suppuration. As to the frequency of osteosclerosis, I[102] have demonstrated this complication 11 times in 113 cases

of chronic caries of the temporal bone (10 per cent.), and 13 times in 96 cases of cholesteatomata (13.5 per cent.)

I also reported a case where the antrum was entirely obliterated by new formation of bone, and the aditus ad antrum ended behind as a blind sac.

We must thank Schwartze for an interesting discussion on osteosclerosis of the temporal bone, especially in those cases in which there has developed an atresia of the meatus as a complication of deep suppuration of the middle ear, as a result of various kinds of traumata, but especially operative interference[118].

Schwartze, indeed, does not decide the question of the cause of the hyperostosis accompaning the atresia, or of the relationship between the two conditions, but inclines to the view that the tendency observed in atresia toward bony hypertrophy is probably the result of chronic dermal or periosteal irritation, caused by the stagnation of the secretion retained behind the stricture.

In regard to the carotid hemorrhages that appear as a rare complication of chronic middle ear suppuration, caused by erosion of the canalis caroticus by caries or cholesteatoma of the temporal bone, the following remarkable case has been reported by Zeroni[119].

The case was a man 29 years old who had died of pulmonary tuberculosis. The hemorrhage from the carotid, that took place shortly before his death, was stopped by tamponing. The post-mortem findings reported by Zeroni are as follows: "The wall of the carotid canal lying toward the middle ear shows a large hole. The top of the canal is entirely absent, and on cranial section, there is found in its place granulations infiltrated with pus. The carotis interna, in its entire course through the carotid canal, is surrounded by discolored, partly purulent masses of degenerated tissue. The wall of the artery is softened, infiltrated and discolored in its posterior and superior parts, corresponding to the holes found, but also extending somewhat beyond their borders. Some white nodules can be seen in the infiltrated parts. On the part of the arterial wall toward the middle ear, there is a perforation, the size of a pin; its position is about the

place where the artery passes from its vertical to its horizontal course."

Although not confirmed by histologic examination, the case according to the macroscopic picture, was one of caries tuberculosa and tuberculous affection of the carotid wall. The place of the arterial arrosion corresponds to Hessler's site of predilection, the convexity of the transition of the artery from the vertieal to the horizontal course.

Zeroni explains the predilection toward perforation at this place by the fact that the vessel lies here nearest to the bony wall, and thus the danger of injuring the tube by a sequestrum caused by tuberculous disease is the greatest. Furthermore on account of the nearest approach of the vessel to the bone, the propagation of the bone disease over to the vessel coats take place here the easiest and the softening process in the vessel walls at this place has usually most widely progressed. In addition, the blood current most easily causes arrosion here, because the blood propelled by the action of the heart through the arterial system, naturally strikes the sharp curve of the carotid, whose convex side receives the greate t force.

A supplement to this case is the one of Heine[120], reported from Lucae's clinic, in which an arrosion of the carotid occurred as the consequence of a cholesteatoma of the temporal bone.

A case of Heermann[121], was one of a girl, 3 1-2 years old, with chronic otorrhea, who died of leptomeningitis. The postmortem showed caries of the promontory, a vestibule filled with pus and granulations and a defect in the floor of the middle ear. The carotis interna lay here free, surrounded by granulation tissue. On the side toward the middle ear was a small perforation in the wall of the carotid, through which a probe could be passed. The carotid throughout its canal, was surrounded by a purulent mass, its wall was discolored and very friable, and its lumen was filled with a thrombus, saturated with pus.

Jürgens[122] has reported two other cases.

In the first case, a recruit who had died of a diffuse purulent leptomeningitis, the right temporal bone was carious to a great extent, as though bitten out, saturated with pus, and

discolored. The walls of the right carotid were necrotic to the extent of 2.5 cm. where they pass over into the horizontal portion. The walls were thickened, discolored and uneven. In the medial part of the artery was a fresh, nonsuppurating thrombus.

The second was also one of a recruit who had died of a diffuse purulent, otogenous meningitis. The right os petrosum showed carious destruction to a considerable extent. The middle ear and the carotid canal formed a large, irregularly excavated cavity. The walls of the artery, in the bony canal, were entirely necrotic, not only at the knee but also almost the entire horizontal portion, almost to the point of the pyramid.

From the extensive literature of the last decade dealing with otogenous paralysis of the facial, we have selected only a few anatomical findings, that show the anatomic substratum of paralysis.

We have long known that in acute otitis media, with a complete filling of the middle ear with exudate, we may have a paralysis of the facial, probably by pressure of the exudate on the trunk of the nerve through a dehiscence in its canal. We must thank Panze[123] for an anatomic finding of this sort. He made serial sections through a temporal bone with acute "tympanitis." Several sections showed a hole in the wall of the canalis Fallopiae. The exudate filling the middle ear touched the perineurium, and in places even invaded the meshes of the fibrous tissue.

Grunert and Meier[124] had the opportunity of showing, in a case that came to postmortem, the anatomic substratum of artificial paralysis of the facial, which was caused by the attempt to remove a polypoid granulation from the facial prominence. The postmortem shows hemorrhages between the nerve fibres.

Of the greatest interest is a case of Buhe[125] from Schwartze's clinic, which shows us the anatomic substratum for a 35 year old facial paralysis. It was the temporal bone of a 35 year old suicide who had suffered from a paralysis of the facial since his first year.

The whole middle ear was filled with porous masses of bone. From the ganglion geniculi ran a very thin band of

the facial, in about the normal position, above the promontory, while the main portion of the nerve, some millimeters below the ganglion geniculi, took a course directly across the middle ear, and ended in the bone of the lateral wall of the middle ear, above and in front of the drum. The lower part of the middle ear was also filled with spongy masses of bone and fibrous tissue. Only in two places were small cavities, which were filled with detritus, one at the mouth of the Eustachian tube, and the other beneath the second knee of the facial, at the region of the round window. The drum was not easily discoverable from within on account of fibrous adhesions. When the paracentesis was made, the needle went through the drum into the fibrous mass that filled out the lowest part of the middle ear. The osicles were entirely absent. When the facial was laid bare, from the direction of the base of brain, it could not be readily found in the mass of connective tissue, and only at its exit at the foramen stylomastoideum was it recognizable as a fibrous band that could be followed into the bone. Since the meatus, on account of the slight development of the processus mastoideus, lay close to the base of the brain, the piece from the exit to the second knee was very short. Just below the second knee, the main body of the facial diverged and ran directly upward toward the base of the brain to end blind in the bone, while a very thin remnant formed the band that continued along the normal course into the middle ear.

This dislocation of the facial, since an injury may be excluded on account of lack of operative interference, can be explained only on the supposition that an exfoliation of the bone, which took place according to the statement of the mother, not only completely tore the nerve trunk but also involved the end of the nerve.

Our knowledge of the pathology of the aural cholesteatomata has been in many ways increased during the last decade, but the question has not yet been settled.

That the earlier view of the pathologic anatomists, who considered cholesteatomata to be true new formations developed from misplaced cells of the ectoderm, is not correct in the majority of cases, but that on the contrary they are etiologically related to the ingrowth of the epidermis of the

meatus or the skin, into a suppurating middle ear, which takes place under certain circumstances, was shown by Habermann two decades ago, but some aurists still hold to the other view.

A certain confusion has probably been caused in the entire cholesteatoma question by the application of the term "cholesteatoma" to the most heterogenous bodies, even to such as do not deserve such a designation.

Here belong the so-called cholesteatomata of the external meatus, which Habermann[126] described, and which correspond to the "masses of epidermis" of Hessler. Although Habermann, in contradiction, to Hessler, recognizes as an etiologic factor the chronic otorrhea which frequently precedes these bodies, and although their structure, growth and the ulceration of the bone hat frequently is caused by them bear a great resemblance to the characteristics of middle ear cholesteatomata, nevertheless we must deny to them the title "cholesteatomata" because they are not found in spaces that do not normally possess epidermis, but mucous membrane.

The so-called cholesteatoma in aural polypi (Habermann, Zeroni, Kümmel) the epithelial pearls of the drum, of unknown origin, as well as those arising in scars of the drum, (Habermann, Grunert) must also be considered, according to our view, as not belonging to the cholesteatomata.

There is no doubt that there are primary cholesteatomata of the temporal bone To the well known case of Lucae, a second has been added in the last decade.

This case of Schwartze[127] was that of a 41 year old man who died of phthisis pulmonum, who had an almost circular tumor, of the form of a biconvex lens (diameter 4.5 cm. by 1 to 1.5 cm. thick), in the squama of the right temporal bone, which reached into the pars petrosa and had ulcerated the squama outward and inward. Internally, the bone curved forward and touched the tumor. The tumor had a lamellated structure with a mother-of-pearl luster of the lamellae. The microscopic examination of the contents of the tumor showed the lamellated arrangement characteristic of the cornification products of the squamous epithelium. The tumor was surrounded by a mantle of connective tissue, poor in nuclei and in places slightly infiltrated with cells, whose inner surface was lined

by several layers of squamous epithelium. In the lower half of the drum was a scar; the hammer, anvil and mucous membrane of the middle ear were normal. As to the pathogenesis of this tumor, it was probably due to a misplacement of squamous epithelium in the period of embryonal development, and the cholesteatoma developed after the analogy of the cholesteatomata observed on the membranes of the brain.

In the case of cholesteatoma verum in the posterior cranial fossa, reported by Körner[128] the temporal bone was affected secondarily by the tumor growing into the pneumatic middle ear spaces.

The literature shows beyond doubt that true primary aural cholesteatomata belong to the rarest of occurences.

Körner[96] in agreement with Kuhn, considers true cholesteatomata of the temporal bone (heteroplastic new formations whose anlage falls in the embryonal time) with secondary suppuration of the ear to be much more frequent than those which arise according to Habermann's idea, as secondary to chronic suppuration of the ear. The latter he calls pseudo-cholesteatomata or, with Wendt, otitis media desquamativa.

We cannot agree with this view which has been abandoned by Körner, and before him by almost every pathologic anatomist, because we have never found a cholesteatoma in the large number of 1 year old children operated on in Schwartz's clinic for mastoid, also never in those cases which were operated for undoubted acute mastoiditis. Although Körner, among other reasons for his view, says that in cases of "pseudocholesteatomata" the epidermal ingrowth "is accustomed to progress exceedingly slowly, and seldom leads to a collection of a considerable mass of epidermis" we can recall some cases of artificial cholesteatomata (published by Panse and Grunert from Schwartz's clinic) in which the first mastoid operation showed not a trace of cholesteatomata in the mastoid, while at the second a more or less considerable collection of cholesteatomatous masses was found. The greater or less amount of epidermal product depends, in our opinion, on the degree of the inflammatory process going on in the matrix (dermatitis hypertrophicans exfoliativa).

THE CHOLESTEATOMATA SECONDARY TO SUPPURATION OF THE EAR.

This includes the great majority of the so-called aural cholesteatomata. The designation "secondary" cholesteatomata can be replaced by cholesteatomata "developed extrauterine." The so-called primary, "true" cholesteatomata are to be designated as "of intrauterine origin." Both are the production of epidermis in parts of the ear that normally do not contain epidermis. The groundwork for this pathologic production of epidermis is, in the one case, the intrauterine migration of epidermis into the middle ear.

(a). Habermann's cholesteatomata.

Steinbrügge was the first to insist that, in addition to the migration of the epidermis into the suppurating middle ear (Habermann) a second factor was necessary, viz,, the inflammation of the new lining of the middle ear. While Steinbrügge thought that the irritation leading to the inflammation of the migrated epidermal lining took place in the lower layers of the epidermis, Haug[129] claimed that the irritation of the pus, that was prevented from escaping, acted on the surface of the migrated epidermis. It macerated the superficial epithelial layers and since the organism attempted to free itself from dead cellular elements, the subjacent epithelial layers were made to proliferate, but were pressed upon by the old layers lying on them and by the masses of secretion, and being pressed back, in their turn became a source of irritation to the following layers. He called this procedure the strife of the epithelium with its offspring.

The roll played by caries of the ossicles in the pathogenesis of the aural cholesteatomata was first pointed by us[102]. The ossicles sometimes form the bridge over which the epidermis enters the middle ear, in cases of high perforations. While Jansen[130] had mentioned the frequency with which cholesteatomata of the middle ear are accompanied by fistulae into the bony, horizontal semicircular canal, which comes about through pressure ulceration, and which often leave the membranous semicircular canal intact, we (l. c.) insisted that in this way cholesteatomata could enter the internal ear. The fistulae allow one to think of the possibility

that in certain cases they may be caused by caries, and that the epithelium of membranous semicircular canal in this way can get into the middle ear and cause the formation of cholesteatomata.

The old view of Kirchner, in regard to the growth of a cholesteatomata, and the origin of the ulceration of the bone, that it was caused by an active growth into the Haversian canals, has lately been proven erroneous by Katz (l. c).

Siebenmann considers the bony rarification under the matrix in cholesteatoma at those places where there are epidermized plates of bone—e.g., the walls of the pneumatic cells in the mastoid—to be analogous to atrophy of the turbinates in ozena. "It seems that the physiologic balance between apposition and resorption is more disturbed at those places where the supply of blood must come from a metaplastic region. It is possible that these vessels suffer greater changes than the few contradicting investigations lead us to suppose."

The spontaneous cure of cholesteatomata has been repeatedly observed. We[102] have shown the fact, hitherto unknown, of the spontaneous cure by calcification of the matrix.

(B) CHOLESTEATOMATA CAUSED BY METAPLASIA OF THE MUCOUS MEMBRANE OF THE MIDDLE EAR.

Before Habermann had shown that the majority of cholesteatomata are caused by the migration of the epidermis of the external into the middle ear, aurists shared in the belief of v. Tröltsch and Wendt in regard to the pathogenesis of of cholesteatoma. They claimed that the matrix of the cholesteatoma arose by a metaplasia of the epithelium of the middle ear's mucous membrane into squamous epithelium, and v. Tröltsch thought that this took place as a result of the irritation and pressure of the stagnant cheesy pus, while Wendt thought it was the result of unknown external causes, which acted on the mucous membrane through the perforated drum.

Although since Habermann's explanation, we have almost

entirely given up the view of v. Tröltsch-Wendt, the last decade has given us some anatomic findings that force us to take that into consideration in certain cases of cholesteatomata.

Politzer[132] has demonstrated that pathologic mucous membrane of the middle ear may undergo an epidermic transformation, which can cause the formation of a cholesteatoma. He possesses a specimen of a smooth cholesteatoma, size of a bean, in the upper part of the middle ear cavity, which took its origin from an obstructed and dilated cyst of the mucous membrane. According to Morf[133], a direct metaplasia of the epithelium of the mucous membrane into the epidermal matrix of a cholesteatoma was observed in Siebenmann's clinic.

The case reported by Dreyfus[134] from the same clinic, of severe tuberculosis of the middle ear that ended in a cholesteatoma, where there arose "the cholesteatoma matrix, with a superficial epidermal layer, in the bony cavity, which was covered by an epidemis membrane, by a change of the most superficial layers of the bone scar in consequence of metaplasia of tissue," cannot be explained in this manner, because it was not a case of metaplasia of the epithelium of mucous membrane into epidermis.

(c) THE CHOLESTEATOMA OF LEUTERT[135].

They have a peculiar position as the connecting link between true tumors and Habermann's cholesteatomata, in so far as they are true retention tumors, which have arisen on the basis of epidermis which has migrated into the suppurating middle ear.

Leutert (l. c.) suggests that the epidermis that has grown into the middle ear is able to cause cholesteatomata not only by over production of epidermal products on an inflammatory basis, but also by actual tumor formation, by forming of retention cysts.

His cholesteatomata form a completely closed sac, and consist of three layers, an external, connective tissue, a middle, squamous epithelium and a central product of cornification of the latter. These retention cysts arise from epidermis which has been severed from its connection either by inflam-

matory processes or by operative procedures, and has grown into a closed sac, according to the character of a traumatic epithelium cyst. The presence of such very small cholesteatomata, which grow by the accumulation of the products of cornification within them, and without the presence of inflammation, on the long process of the anvil, in connection with centrally lying perforations, and without an epidermal path leading to them, causes Leutert to assume a further possibility for the origin of this kind of cholesteatoma, viz. the transplantation of pieces of epidermis from the drum into the granulation tissue lying behind it by means of instruments, etc. The growth of these implanted pieces of epidermis takes place exactly analogous to that of traumatic epithelial cysts, and the processes in Kaufmann's encatarrhaphia.

While we have just shown that the study of the pathogenesis of cholesteatomata has received, during the last decade, an appreciable widening and deepening, the same space of time has brought us a large number of remarkable cases

Hugel[136] reported a case in which the formation of a cholesteatoma continued in the middle ear in spite of the cicatrization of the drum.

I have seen a similar case in our clinic. In a case where there was a differential diagnosis to be made between abscess or gummi in the temporal lobe, on the grounds of the cicatrication and calcification of the drum, I diagnosed the latter, but the autopsy showed an otitic abscess of the temporal lobe caused by a cholesteatoma in the recessus epitympanicus.

In regard to the spreading of the cholesteatoma, the following are of interest.

Grunert and Meier[124] reported a case in which the autopsy showed an extensive cholesteatomatous focus in the region of the apex of the os petrosum to be the cause of the continuance of a suppuration after the complete cure of a large cholesteatoma of the middle ear.

I[102] reported a case of a cholesteatoma, the size of a cherry, under the surface of the bony inferior wall of the meatus, and a second one, in which the cholesteatoma, after destroying a large part of the anterior bony wall of the

meatus, had pressed into the fossa glenoidalis of the lower jaw. Kretschmann[137] reported a case of arrosion of the wall of the sinus sigmoideus by the mechanical pressure of a cholesteatoma.

In the case of Lubet-Barbon[138], the temporal bone was changed into a cholesteatomatous cavity of the size of a hen's egg, which was surrounded only by a soft shell, accompanied by complete disappearance of the bone beneath the dura, sigmoid sinus, and periosteum.

In the case of Haug[139] the cholesteatomatous masses had pressed up to the lateral ventricle. The whole middle ear was filled with the cholesteatoma. Just at the sinus sigmoideus, which was filled with a thrombus, the dura was perforated to the size of a pfennig, and the vicinity of the perforation was greyish green discolored. The bare surface of the brain was greyish green discolored, and in an area of the size of a 20-pfennig piece, was eaten out by the cholesteatomatous masses that had broken through. From this area, the semisolid cholesteatoma, in places mixed with blood, and surrounded by softened masses of brain substance, extended to the left hemisphere of the cerebrum between the gyrus fusiformis, hippocampi, gyrus lingualis, along the interstices of the vessels, i. e., between the temporal and occipital lobes, into the inferior and posterior brain. In the lateral ventricle was a foreign body consisting of masses of cholesteatoma.

Finally, we must mention a case of Barnick,[140] in which a cholesteatoma of the mastoid was changed into a cyst containing blood. This change was caused by a hemorrhage from the arroded sinus or a large bone vessel into the cholesteatoma cavity.

In concluding the findings of the last decade concerning the bone complications of otitis media suppurativa chronica, we must mention, on account of its rarity, a case published by Sonntag,[141] of the residues of chronic suppuration.

"After the removal of the normal tegmen tympani, the head of the hammer and the body of the anvil appeared in the attic in the normal manner; there was lacking, however, the connective tissue connection between the head and the body of the anvil. These were firmly bound together by

bony tissue. A shallow furrow was the only indication of the joint between the two bones. From the hammer-anvil body a broad bridge of bone, perforated by a point-like opening, ran to the mesial wall of the middle ear. The anterior wall and the anterior part of the mesial wall of the middle ear were connected with the above bony bridge and the neck of the hammer by a broad band of bone, which passed upward in a continual mass of bone, in which the handle of the hammer and long process of the anvil seemed to be enclosed. These broad bridges of bone prevented a further investigation of the middle ear from above". After the middle ear was opened f om below, "a mass of bone was seen which occupied the entire breadth of the middle ear except a small hole Neither in the bone nor in the cavity were there to be found single items such as the end of the long process of the hammer, the stapes, the cochlear window, or the promontory. The impression was given that the entire over-half of the middle ear was filled out with one mass of bone and that out of this the head of the hammer and the body of the anvil were the only things that projected in unchanged form. The mucous membrane that covered the mass of bone was thickened." As the mesial bar of the normal meatus there was found, instead of the drum, a bony plate of the size and position of the drum which in its anterior inferior quadrant was perforated by a small hole. A straw introduced into this hole appeared in the middle ear just before and beneath the head of the hammer. While the pars externa of the attic had remained free from the new formation of bone, that which filled out the lower half of the middle ear extended in one mass from the region of the wall of the promontory to the plate of bone which closed off the meatus. No new formation of bone was found in the rest of the middle ear.

If we turn to the findings of the last decade concerning tumors of the middle ear, we must say at the outset that the discussion of polypi and the cholesteatomata that are not to be included in the true neoplasms has already taken place, because we must give them a special classification as opposed to tumors of the middle ear, on practical grounds if not theoretical.

To characterize the findings of the last decade in a few

words, they consist of new conclusions in regard to the pathogenesis of carcinoma of the middle ear, drawn from a rich material, in addition to the report of the presence of a chloroma in the temporal bone, which had not been observed before.

As to the most important of the conclusions in regard to primary carcinoma of the middle ear, the greatest attention must be paid to a work of Zeroni[142], who has worked over this subject by the aid of the special literature and a number of aural carcinomata at Schwartze's clinic.

He emphasizes the etiologic relation between chronic suppuration of the middle ear and primary carcinoma of the middle ear. The fact that those cancers which certainly take their origin from the middle ear are all squamous epithelium cancers. while in the normal ear there is no squamous epithelium, he explains by migration of squamous epithelium into the middle ear in cases of chronic suppuration just as it is antecedent to the formation of a cholesteatoma. By convincing arguments he repudiates the hypothesis of metaplasia of the cylindrical epithelium of the middle ear which has been proposed, as well as the hypothesis of abnormal development of germinal tissue as the point of origin of the primary carcinoma of the middle ear. Likewise he refutes the theory of Kretschmann and Kuhn, that the cancer possibly comes from aural polypi with many layers of epidermis, by pointing to the histological differences between malignant, atypical epithelial carcinomata and the benign, atypical epithelial growths, which are frequently found in aural polypi, in chronic suppuration of the middle ear. Furthermore, he describes the secondary changes brought about in the different parts of the temporal bone by the growing tumor. A part of the bone is caused to disappear by the direct pressure on it, and a part is made necrotic, with the formation of sequestra, by disturbances of circulation in the bone. The sites of predilection of bone destruction are the walls of the meatus, the temporal squama, the tegmen antri and tympani; on the other hand, as especially resistant he mentions the hard, bony parts of the pyramid, and especially the cochlea. Finally, he describes the varying power of resistance of the dura to cancer, as well as the destruction of

the lateral wall of the sigmoid sinus, and the extension of the tumor into the sinus, which almost invariably takes place.

We must here refer to one of his cases (from Schwartze's clinic), although it was a case of carcinoma of the concha and not of the middle ear. The infiltrated lymph nodes in the vicinity showed a tuberculous disease instead of a carcinomatous. The carcinomatous ulceration must have been the place of entry of the tubercle bacilli; they were then taken up by the lymph nodes, and a local tuberculosis of lymph nodes was caused. In this case we have a new example of the falseness of the belief in the antagonism between carcinoma and tuberculosis, which was formerly accepted.

In the cases of squamous epithelium cancer of the middle ear of Sturm[143], from Körner's clinic, and of Alexander[144], the carcinoma likewise arose on the basis of a chronic suppuration of the middle ear. In both cases the squamous epithelium that laid the groundwork for the cancer had grown into the middle ear from the external meatus, in Sturm's case through a fistula in the superior posterior wall of the meatus. In both cases, the origin of the carcinoma is analogous to the origin of squamous epithelium cancer in old bony fistulae, which according to v. Volkman arise from the epidermis of the skin that gradually grows into the fistula. We might mention another point common to the squamous epithelium cancers which arise on a basis of chronic suppuration of the middle ear and Volkmann's carcinomata in old bony fistulae; viz., both show a relatively slow growth and scant tendency toward metastasis.

In Sturm's case, the carcinoma had completely grown through the temporal bone and had extended to the middle and posterior cranial fossae, the labyrinth and the fossa glenoidalis. In the horizontal part of the sinus transversus was a gray softened thrombus, while the entire vertical part was involved in the tumor.

As to the individual cases, we must mention, on account of its rarity, a case of chloroma of the temporal bone which was observed by Körner[145], and historically examined by Lubarsch[146].

The case was in a 6-year-old boy with multiple chloromata, in part symmetric, of the dura mater, the sinus trans-

versi, the middle ear, the orbit, the temporal musculature, and with subepicardial and subpleural hemorrhages, hemorrhages into the lungs, dilatation of the ventricles of the heart, general anemia, and hypertrophy of different lymph nodes, especially those of the neck. The histologic examination showed that the majority of the cells were of the lymphocyte type, and the Biondi stain showed a fine fibrous reticulum. As a whole, the histologic structure agreed with the reports of other authors as to the minute structure of chloroma. We must especially mention certain structures found in the new formations in the ear, the sinus transversi and the dura; (1) large round cells, with two vesicular nuclei; (2) cells with 4 to 5 nuclei "that might be called giant cells." The chloroma was partially in nodular form, and partially of a more diffuse, infiltrating form, as, e. g., in the bones of the cranium, in the musculus temporalis, and in the orbit. As to the location of the tumor in the ear, the middle ear was entirely free, while tumor masses were found in the mastoid, the subjacent bone of which in places showed a beautiful osteoplastic layer, and in others Howslip's lacunae with osteoclasts. The acusticus was imbedded in a tumor mass, which, however, reached only to the perineurium. Likèwise there was partial degeneration of the nerve fibres (Weigert's stain for medullary substance). Lubarsch, in agreement with v. Recklinghausen, classes chloroma among the leukemic and pseudoleukemic lymphomata and thinks it very probable that in the etiology of lymphosarcomatosis and kindred diseases, an important role is played by the tubercle bacilli and their toxines.

Among the cases of carcinomata reported in the last decade, the following deserve notice:

1. The case of Krepuska[147]. The tumor arose from the glandulae ceruminiferae and extended as far as the middle ear. The hammer was changed into a connective tissue structure, bare of bony elements, and filled with cancer nests; the anvil, completely imbedded in the cancer, showed only in places, on its mucous coat, a carcinomatous degeneration.

2. The case of Haug[148]. The primary seat of the endothelial cancer was in the middle ear. From here, the tumor

had grown downward and inward along the tube, and upward along the hiatus subarcuatus. In contrast to the usual tendency of cancer to break through everything the new formation had advanced along the performed routes, without at first attacking the bony walls; only later when the latter were so much compressed that they could no longer allow expansion, was the bone itself affected; on the base of the brain the tumor had advanced as far as the foramen jugulare and the anterior condyloid foramen. It is interesting to note the length of time that elapsed—at least 4 years—before death, which took place from a terminal meningitis.

3. A case of Schwabach of a squamous epithelium cancer. The seat was in the sphenoid, but it extended over a large area of the base of the brain, and had secondarily attacked the middle ear, into which it had grown in the form of a cone like projection of the floor of the middle ear, lying in front of the niche of the round window. The exact histologic finding was as follows: "The bone of the promontory was intact. The mucous membrane of the middle ear over the tegmen tympani was partially carcinomatous infiltrated; partly entirely destroyed. That of the labyrinth wall was for the most part well retained, even the epithelium being unaltered. The mucous membrane over the promontory showed new formations, and the changes can be followed from here, in front of the niche of the round window, to the floor of the middle ear, where the affection of the mucous membrane is the plainest. From the promontory, the tumor has grown into the middle ear in the form of a cone, and in places fills the entire space as far as the drum. The niches of the round and oval windows are likewise almost entirely filled with this growth, and the ossicles are imbedded in it with the exception of the head of the hammer and a part of the manubrium. This is especially true of the stapes-anvil articulation and the bars of the stapes. The drum is for the most part unaltered, showing only a thickening of its layers, but is not attacked by the carcinoma except at a small place at the superior posterior part, where the tumor had pierced the drum and grown into the meatus, without attacking the walls of the latter. In the cellular spaces of the mastoid is a rather rich fibrino-purulent exudate. The upper wall of the Fallopian canal is destroyed by the tumor, but

the sheath of the facial nerve is completely intact. Likewise, the nerve fibres are seen to be free from pathologic changes both here and throughout the entire course in the temporal bone.

NOTEWORTHY CASES OF SARCOMA.

1. A case of Haug[150]. Melanotic giant celled sarcoma of the meatus, middle ear, and the pars squamosa of the temporal bone which had started in the periosteum of the middle ear.

2. Story[151]. Sarcoma of the middle ear, which appeared under the form of an ear polyp, which quickly returned after removal.

3. Ash[152]. Case of myxosarcoma, melanosarcoma and myelosarcoma of both mastoids, with multiple foci else where.

4. Grunert[153] (Schwartze's clinic). Case of sarcoma of the base of the skull in a three-year-old girl, with perforation of both pterygoid processes of the sphenoid, extension to the temporal bone, the right orbit, metastases with destruction of the bony substance of both frontal eminences.

5. Kirchner[154]. Myelogenous sarcoma in a man, aged 40 years, starting from the cells of the processus mastoideus. It possessed the peculiarity that the spindle cells of the sarcoma grouped themselves in concentric layers around the large cells of the medulla of the bone, forming a structure that recalled a cholesteatoma.

6. Kuhn[155]. Myxosarcoma, which took its origin from the connective tissue on the labyrinthine wall, where myxomatous tissue is found in the new born, and frequently in the first months of life. The soft, greyish red tumor, of the size of a nut, had grown out of the middle ear as far as the external opening of the ear, beyond which it protruded, had perforated the lower wall of the meatus to a considerable extent, was lobulated at its peripheral end, and extended as far as the insertion of the m. sternocleidomastoid (just under a suppurating fistula in the skin over the tip of the mastoid). The mastoid was free from pus and tumor masses. Death of the 1-year-old child, five months after the first appearance of the tumor, of marasmus.

7. Friederich[156]. A sarcoma arising from the dura and involving the middle ear.

8. Schmeden[157]. A small alveolar sarcoma, which had involved the pars pyramida—except the labyrinth—and the mastoid. The entire bulbus venae jugularis was likewise affected and obliterated. In the region of the fossa jugularis, the tumor was as large as a walnut, and superiorly it had grown through the foramen jugularis into the cranial cavity, to the s ze of a large hazelnut.

9. Hegener[158] (Heidelberg clinic). A case of alveolar endothelioma of the right temporal bone, in a boy three years old, wh ch possibly started in the dural covering. The endothelium of the lymph and blood vessel as well as the connective tissue had taken part in the new formation.

Second case. Alveolar melanosarcoma of the right ear in a woman, aged 44, the origin of which likewise could not be determined with certainty.

OTHER TUMORS.

Eschweiler[156]. A case of fibromyxoma of the mastoid; a pedicled tumor of the size of a small egg, which, as well as a part of a cavity formed by antrum, middle ear and bony meatus, was cover d with epidermis.

Haug[160]. A case of gummi of the mastoid region, which developed 8 years after the luetic infection.

Moure[161]. A cavernous angioma, arising from the middle ear. The central cavity was the size of a small hazelnut. The external surface of the tumor consisted of stratified squamous epithelium, with a rete Malpighi below, and well developed papillae in places. Then came a thin layer of connective tissue. This was partly succeeded by irregularly arranged islands of long, dense connective tissue, and partly by an unmistakable cavernous tissue.

Green[162]. A case of osteoma. A lobulated tumor of 15.11 mm. arising from the middle ear, and referred by the author to a remnant of fetal cartilage. Histologically, the periphery of the tumor showed a thin layer of cartilage, which was absent only at the point of insertion, where there was a small pedicle. Under this was a thin layer of compact bone, and under this spongy bone.

Jansen[163]. A hard knobby tumor of the size of a pea, which had destroyed the inner surface of the superior posterior quadrant of the drum, otherwise intact, of a woman 40 years old. There was a striking similarity to the structure of the thyroid gland—areas of cubical epithelium with an alveolar structure and formations of cysts, with colloid substance in the cysts. Nevertheless in the discussion that followed the demonstration, the tumor was regarded as an adenoma and not a misplaced struma.

We have already referred to a portion of the findings of the last decade in regard to tuberculosis of the middle ear, in the general part of this discussion.

Thanks to the pioneer workers, especially Habermann, the pathologic anatomy and general pathology of middle ear tuberculosis is more thoroughly known. Barnick has shown from the material in Habermann's clinic that the tuberculosis of the ear may be caused by a hematogenous infection, as well as the usual method of infection by way of the Eustachian tube, and that there are two forms of tuberculosis of the ear, an acute and a chronic, which must be differentiated. There are still many questions to be solved, e. g., whether in the hematogenous infection, the first development of the tubercle bacilli takes place in the mucous membrane or the bone.

While Körner[96] has lately attempted to differentiate clinically the tuberculosis of the temporal bone, having different origins and courses, we think that the anatomic investigations would also be more valuable, if they would be classed more than they are under the clinical pictures, the more so as certain anatomic groups actually are in accordance with the corresponding clinical types.

Körner's division is as follows: (1) tuberculous suppuration of the ear and temporal bone in the end stages of tuberculosis of the lungs; (2) tuberculous suppuration of the ear and temporal bone where the pulmonary tuberculosis is stationary; (3) tuberculous suppuration of the ear and temporal bone preceding the tuberculosis of the lung; (4) a typical tuberculous suppuration of the ear and tuberculosis of the temporal bone; (5) tuberculous infection of the middle ear and the temporal bone by way of the tube, arising from a pre-

existing tuberculosis of the nose; (6) tuberculosis of the temporal bone by infection via the blood stream; (7) tumor form of tuberculosis of the ear and temporal bone.

There is, as yet, no agreement among specialists, as to the answer to the elementary questions, how often suppurations of the middle ear are of tuberculous nature, and how often a suppurating middle ear is to be regarded only as the port of entrance of a tubercular secondary infection.

In regard to the first of these questions especially, authorities are exactly opposed to each other. The surgeons frequently maintain that the chronic suppurations of the middle ear in the majority of cases have a specific tuberculous basis; on the other hand, the influence of tuberculosis in causing suppuration of the middle ear is minimized by some. That the investigations, hitherto carried on to solve this question, have not yet produced incontrovertible results, is due as much to the fact that the number of investigations at our disposal is still too small to settle it, as to the fact that usually the investigator has confined himself to the bacteriologic examination of the aural discharge. the negative result of which is no proof that it is not a case of tuberculosis.

Under the circumstances, all new, correct statistics are of value.

Brieger[146] found in 241 patients that died of tuberculosis, 37 times chronic inflammatory processes in the ear, whose tuberculous nature he was able to demonstrate 18 times. In 7 cases the tubercular process had attacked the labyrinth; the perforation from the middle ear took place 3 times through the round, twice through the oval, and twice through both windows.

Garbini and Ballistreri[165], in 40 cases of chronic suppuration of the middle ear and caries, could demonstrate 4 times the tubercular nature of the disease by means of bacteriologic examination and experiments on animals.

Körner[166]. A case of tuberculosis of the temporal bone, in which the thickened dura mater, covered with granulations, lay bare over the destroyed tegmen tympani. The granulations contained pieces of bone; the dura, as well as an adhering piece of brain substance was permeated by caseated tubercle nodules.

Hanel[167]. A case of beginning perforation of both windows of the labyrinth in caries tuberculosa of the middle ear. The invasion of the labyrinth by tubercular disease took place at both windows. "The periosteal inner layer was laid bare at the round window by caries of the bone, in such a way that the fibres of the membrane of the window had lost their insertion in the carious bone, and thus nothing was in the way of the granulation tissue growing under the endothelium of the membrane of the window and the periosteum of the labyrinth." "In the anterior, inferior periphery of the oval window, however, the comparatively thin ligamentum annulare was itself destroyed by the caseation that had advanced over the surface. Here the thickened endothelium layer of the ligament still offered obstacle to the process, so that there was really no perforation with free communication between the perilymphatic space and the middle ear."

Grunert and Meier (l. c.), A case of tubercular panotitis, from Schwartze's clinic. in a two year old child. Even the macroscopic findings gave the diagnosis of tubercular disease, which was confirmed by the microscopic examination (tubercles in the granulation tissue, tubercle bacilli in the aural discharge). The mouth of the external meatus was widened by ulceration (11.9 mm.). Anteriorly, the ulceration, which had destroyed the tragus, passes over into an ulcer of the skin, 3, 5:1.3 cm. in size, with undermined margins. The ear was changed into a cloacum the size of a pigeon's egg. None of the membranous meatus was to be seen, and of the bony walls, the anterior and posterior were entirely destroyed, and the superior and inferior were almost entirely so. None of the anterior attic wall remained. Posteriorly, all of the processes mastoideus was lacking except the tip, so that the posterior wall of the cavity was formed by a portion of the sinus transversus 2 cm. long. A part of the Fallopian canal was found in a bony sequestrum. The macerated temporal bone showed, futhermore, a large defect which started in the labyrinthine wall, in the region of the oval window that was suppurating and carious, crossed the entire pyramid of the temporal bone in an oblique direction, and joined with the meatus internus. Furthermore there

was an extensive carious erosion of the posterior semicircular canal throughout its entire extent.

Barnick[168]. A case of acute tuberculosis of the middle ear in a female aged 32—7 weeks from the beginning of the ear affection to exitus lethalis. There was a "high degree of diffuse edema and infiltration of the mucous membrane of the bony tube and middle ear. On the lateral wall of the labyrinth was a superficial ulceration and caseation of the mucosa, while in the deeper layer were well characterised miliary tubercles with giant cells. The bony walls of the middle ear were not affected. Both membranes were entirely destroyed. Labyrinth intact."

The histologic findings in a case of a woman aged 20, who died of chronic tuberculosis of the middle ear were as follows: " In the bony part of the tube, as well as in the part of the middle ear near its mouth, a superficial caseation of the mucous membrane with numerous miliary foci and characteristic giant cells in the deeper layers were found. The mucosa of the attic, the antrum, and the internal and inferior walls of of the tympanic cavity were completely caseated, and the adjacent bone was partially eroded. The long process and the body of the anvil, the manubrium mallei and the anterior crus of the stapes were carious, and the latter was fractured by granulation tissue, containing tubercles. The tubercular growth had completely destroyed both labyrinthine windows, although there was nowhere a perforation, but there would certainly have been one in a short time at the poste ior inferior border of the body of the stapes. The facial canal was likewise arroded over the oval window, and its sheath was inflammatorily thickened. The labyrinth was free from all signs of inflammation."

Piffl[169]. An interesting anatomic finding of otitis tuberculosa, with tumor like protuberance into the cranial cavity, from the pathologico-anatomic institute of Prof. Chiari at Prag. Extensive tubercular destruction, which had extended by several paths from the middle ear into the internal ear. The disease finally passed over into the cranial cavity, caused a pachymeningitis externa tuberculosa, and then a reactive inflammation of the dura mater over the attic, which lead to a tumor like thickening of the same. "In the angle

between the superior surface of the pars petrosa and the internal surface of the pars squamosa, was found the main mass of a 'tumor" larger than a walnut, whose surface was covered with a large number of nodules varying in size from a millet seed to a hemp seed." This tumor belonged exclusively to the dura. Similar large tumor-like thickening of the dura on the posterior surface of the petrous pyramid, tumors in places 2 cm. thick, growing around and obliterating the sigmoid sinus, obliteration of the bulbus venae jugularis. The tubercular nature of the tumor masses was confirmed by histologic examination (tubercles, central caseation, giant cells, tubercle bacilli).

Warnecke[170]. A case of cholesteatoma with tuberculoma of the cerebellum and meningitis tuberculosa. It was not shown in this case whether the diseased ear was the point of entrance of the tubercular infection of the cranial contents or not.

Haike[171]. A case of isolated caries tuberculosa of the labyrinth wall in a patient that did not show tuberculosis clinically.

Bär[172]. The results of the histologic examinations of tubercular granulation-tumors, in two cases from the ear clinic at Strassburg.

The granulation taken from a retroauricular abscess cavity showed the histologic structure of tubercular granulation-tumors, the so-called granuloma. The same appearance was given by the aural polypi present. The latter were probably ordinary polypi, that had secondarily become infected by tuberculosis.

Liaras[173]. A case of lupus of the middle ear, in which a proliferation of connective tissue caused a complete obliteration of the middle ear and the Eustachian tube.

In regard to no portion of the pathologic anatomy of the middle ear have our views undergone such a radical change in the last decade as in regard to the so-called sclerosis of the middle ear.

Although a decade ago, the middle ear sclerosis of the so-called dry chronic catarrh was a very vague conception under which were grouped pathologic conditions of the most varied anatomic basis, at the present time, thanks to the labors of

Politzer, Bezold, Panze, Habermann, Ed. Hartmann, Siebenmann, and Katz, the pathologic anatomy of that disease has been so well determined, that we know the anatomic conditions characteristic of it. It is true that the relationship of the findings to the pathogenesis is still in dispute, and the future must make this clear. The work necessary for this must chiefly consist in clinical observations; less can be expected in this connection from anatomic investigations. It is especially necessary to construct the family trees of otosclerotics, and to give the most careful attention to the constitutional conditions of the individual members.

No better review of the pathologic anatomy of sclerosis can be given than by reporting in a historical form the different steps by which our knowledge has advanced.

Although for decades through the anatomic investigations of Toynbee, v. Tröltsch and Schwartze, we have known the important pathologic changes that underlie the well known clinical symptom-complex of sclerosis, e. g., the new formation of bone in the region of the labyrinthine windows leading to ankylosis of the stapes, the greatest stress was laid on the chronic inflammatory changes in the mucous membrane of the middle ear. Politzer[174] was the first to break away from this view, and, on the basis of 16 cases anatomically examined, claimed that there was a circumscribed, primary disease of the bony labyrinth capsule in the region of the niches of the windows, which caused a new formation of bone tissues and finally complete ankylosis of the stapes. The results of his investigations he summarized as follows:

"The bony tissue of the labyrinthine capsule forming the neighborhood of the oval window is changed into a newformed bony substance. The newformed masses of bone stain much deeper with carmine than the neighboring normal bone. The differences in the color is so pronounced that it is possible to distinguish the pathologic part from the normal with the unaided eye. The bone corpuscles appear more numerous in the pathologic than in the normal bone. But the chief point is the enormous dilation of the bony spaces, which distinguishes the normal from the pathologic tissue. The contents of the space varies in the different cases. In many specimens they are filled with a connective tissue rich

in cells, which encloses larger or smaller blood vessels. In the cases of new formation of bone of later date, the dilated spaces are filled with extensive blood vessels which are laden with blood corpuscles. On the other hand, the older pathologic bony tissue contains in its bony spaces blood-vessels crossing each other in every direction, and containing few corpuscles. Finally, I found in several specimens, in addition to the blood vessels giant cells in varying number, osteoblasts and osteoclasts."

Politzer especially emphasizes the fact that the most apparent changes in the bony tissues are found not in the vicinity of the middle ear mucous membrane, but in the deeper layers of the labyrinth capsule, as well as the appearance of isolated islands of pathologically altered bone in the vicinity of the internal meatus and cochlea, and claims that is due to a primary disease of the labyrinthine capsule.

Although the fact that the cases examined by Politzer were all of advanced age, allowing the inference that they were senile changes, his findings were later confirmed by all other investigators.

In the same year, Bezold[175] explained according to Politzer's theory a case of bilateral stapes ankylosis, in which, especially on the left side the pathologic process was confined to the stapes and its vicinity. Bezold based his explanation on the fact that the histologic examination, especially of the left ear, showed a complete normality of the middle ear (mucous membrane, etc).

Although Politzer's description of the histologic findings, corresponds on the whole, to the anatomic picture of otosclerosis, and although it is apparent from his description that he saw the foci of spongification in the labyrinth capsule, still Siebenmann and his pupil, Ed. Hartmann[176] were the first to regard these foci as something so peculiar to otosclerosis that Siebenmann[178] wished to call the entire process a "progressive spongification of the labyrinth capsule."

The additional microscopic findings with which Ed. Hartmann made us acquainted from Siebenmann's clinic are the following:

"Bony stenosis of the pelvis ovalis, complete ossification of the ligamentum annulare, formation of a spongy bony

wall around the oval window, on the vestibular side, pressing inward of the posterior and pressing outward of the anterior part of the stapes.

On the right side are found similar conditions, with the difference that the most superior part of the upper circumference of the ligamentum annulare is not yet ossified. The spongifying process extended over a large part of the bony cochlear capsule. The cochlear spiral consisted, abnormally, of compact bony substance, which had increased at the expense of the bony and nerve canal. The microscopic examination of the right labyrinth showed, furthermore, a growth of the perineural and perivascular connective tissue in the basal turn, with a normal (?) number of ganglia and nerve bundles and normal organs of Corti. The stapedius muscle had degenerated to a connective tissue band, while the tensor tympani was normal."

In the second case of Ed. Hartmann, the disease was also not confined to the oval window and its vicinity. The pathologic process in th bone of the window margins in both cases had progressed forward as far as the cochlea. The appearence of the bone corpuscles, which in the pathologic bone were plump, irregular, and numerous and dense especially at the places where they bordered on normal bone, speaks for the fact that it was case of new formation of bone and not a conversion of old. Considering the large number and dense arrangement of these bone corpuscles, especially at the places bordering on normal bone, it seemed to Hartmann, that it was probably a new formed, metaplastic, connective tissue bone.

In two cases of ankylosis of the stapes, specimens of which were demonstrated by Habermann[177] at the 7th German Otologic Congress, the ankylosis also was complicated by a disease of the bone extending to the vestibular wall.

One year later, S ebenmann reported a case of the highest interest both clinically and anatomically.[178] In it were found formations of osteophytes on both the tympanal and vestibular side of the oval window, and spongy foci in the bony capsule of the semicircular canals, vestibule and cochlea. In the right labyrinth were six foci of spongification: Only a loose connection existed between the first two of these,

while the others were marked off from each other. Furthermore, there was a beginning ossification of the cartilage of the stapes, with a beginning ossification of the ligamentum annulare stapedius (on the left side). The spot of predilection of the foci of spongification, the oldest of which was found at the border between the primary capsule of the labyrinth and the parostal connective tissue bone, was where the well known remains of primary cartilage are found (Manasse). This fact brought Siebenmann to the conclusion that sclerosis was the last phase in a process of development that seldom appeared in the cochlea.

Siebenmann[180] described another important and complete anatomic finding in a female, aged 64, who died of carcinoma hepatis. Although the contents of the middle ear were normal, there was a single wedge-shaped focus of spongification in the labyrinthine capsule, whose position corresponded to the whole upper border and the anterior half of the oval window. The base of the wedge, 4 mm. in diameter, reached superiorly to the facial canal and the canalis pro nervo utriculo-ampullari as well as to the lateral portion of the canalis pro tensore tympani; its apex lay 2 mm. under the niveau of the inferior border of the fenestra ovalis, in the vestibular wall on the side of the middle ear. Superiorly, the focus extended rather deep into the bone, between cochlea and vestibule. Over the part of the spongy focus that lay immediately under the mucous membrane of the middle ear, was an osteophytic growth of the superior border of the niche, so that the superior border of the stapes plate was hidden externally by an overhanging wall of bone and was pressed somewhat inward. The stapes was bound to the exostosis by a hard band, poor in nuclei; from its anterior border, two osteoid adhesions, only very slightly ossified, extended to the border of the oval window. The inferior part of the plate of the stapes was pressed toward the tympanum, and at the same time dislocated anteriorly and inferiorly. In the few large medullary spaces that still remained in the already old spongy focus there was true medullary substance, a phenomenon not yet observed. Siebenmann explained the acute interstitial neuritis of the trunk of the acoustic nerve as a sequence of carcinoma cachexia.

Siebenmann's theory that the "progressive spongification of the labyrinth capsule" was the last phase in a developmental process that seldom appeared in the cochlear capsule was opposed by Katz[181]. He recognized fully the importance of Siebenmann's anatomic finding of the formation of medullary spaces, but regarded the origin of the foci of spongification as well as the new formation of bone in the sclerosis as always of inflammatory nature, viz. a periostitis of the middle ear.

The anatomic specimen, on which the theory of Katz rests, shows the following points of interest:

"1. The hammer and anvil are extremely osteoporotic, the corresponding joints greatly involved, as well as the attic wall and a part of the neighboring wall of the external meatus.

2. There is pathologic formation of medullary spaces, of an old date, in the labyrinth capsule, especially at the anterior portion of the oval window, and extending from there to the basal cochlear turn or porus acusticus internus, and to the semicanalis tensoris tympani and to the facial canal.

3. An osteophyte on the promontory which narrowed the niche of the oval window, and disturbed the free movement of the stapes.

4. No bony synostosis of the stapes-vestibular articulation. The plate is almost intact, as is the symphysis.

5. The characteristic stain with carmine or hematoxylin of all the places affected by the formation of medullary spaces (resorption and apposition).

6. In the middle ear (attic) was a large amount of fibrous connective tissue which has grown fast to or merged with the mucous-periosteal lining, which is a certain sign of a previous inflammation of the middle ear.

7. Atrophy of the nervus cochleae within the lamina spiralis ossea in the lower cochlear turn. Corti's organ, in spite of allowance to be made for poor conservation, can be regarded as affected. Ganglion cells intact."

The author considers the most important parts of these finding to be the fact that there is no ankylosis, at least no bony ankylosis of the stapes; that the bone disease was not limited to the labyrinth capsule, but extended to the ham-

mer and anvil, and that there were signs of a previous inflammation in the middle ear. Katz (l. c.) in opposition to Siebenmann, regards the bone changes in "sclerosis" as of inflammatory origin, the product of an ostitis vascularis chronica ossis temporum, which he identified with the "vascular ostitis" of R. von Volkmann. He bases his view on the importance of the Haversian canals and the blood vessels running in them in the formation of medullary spaces, as is seen most plainly in individual cases. "In fresh cases I have especially noticed the new formed, wide capillaries in the microscopic examination of the affected bone."

This process in the temporal bone, which is characterised by resorption, without suppuration, of old bone and apposition of a new bony tissue that is less dense and partially more voluminous, he thought he could identify, on the grounds of his microscopic examination, with the chronic vascular ostitis of Volkmann, because in the last d sease "new canals with vessels are formed from the Haversian canals, which break through the lamellae in various directions thus forming medullary spaces, and which finally lead to disappearance of the bone and osteoporosis."

Katz thinks that the undoubted heredity of the affection does not rest on an inheritance of an anatomic abnormality of the temporal bone (narrowness of the Eustachian tube, or the middle ear, according to v. Tröltsch), but on the inheritance of a constitutional diathesis, and that the otitis media was only the accidental condition that started the trouble.

He regards the following dyscrasic diseases as having etiologic relation with sclerosis. 1. The rheumatic-gouty diathesis. 2. The scrofulous. 3. The syphilitic. 4. Unknown senile changes, on which Schwartze lays especial stress. 5. Neuroparalytic or trophoneurotic diathesis.

The demonstration by Katz[182], of a rarefying ostitis of both outer ossicles in a one year old child suffering with chronic otorrhea, causes the thought that a sclerosis may follow a chronic suppuration of the middle ear, or accompany it. And it is true that certain clinical facts show that a sclerosis may be hidden by a chronic middle ear suppura-

tion—it must be left to the future to determine whether the latter is the primary or only the accidental disease.

Although, in general, after the cure of a chronic suppuration, the functions remain stable, we sometimes see in these cases, e. g., in healed radical operations, an increase in the deafness, without any change in the objective findings being demonstrable as an explanation for the fact.

Whether a sclerosis is also at the bottom of this phenomenon must be determined by further anatomic investigations.*

While it seems that an otosclerosis may be masked by other diseases of the ear, we also think that sclerosis may give to other diseases a definite character. To give an example, it seems that a sclerosis is the basis of the cases of acute catarrh "with prepondering hyperemia," to which v. Tröltsch and Schwartze have given a special classification, on account of their unfavorable prognosis.

Furthermore, Habermaun[183] showed that the bone disease begins where the larger or smaller periosteal vessels enter the bone and that it advances in the bone along these vessels.

What conclusions can be drawn as to the anatomic picture of otosclerosis, from the findings of the last decade.

Pathologico-anatomic, the following has been shown; the disease appears in multiple foci. They are usually distinct from one another. Only in one case (Siebenmann$_{178}$) was there a loose connection between two foci. The appearance of these foci is irregular, in one case (Siebenmann,[180]) it was wedge shaped. These foci are found especially in the labyrinth capsule usually near the window, where they generally lead to ankylosis of the stapes—exception, Katz[181]. They appear, however, also in the bony portions of the cochlea, and may lie outside of the labyrinth capsule, e. g., near the internal meatus.

According to Siebemann$_{178}$ they are prone to occur where the well known remains of the primary cartilage are found (Manasse). But they also are found outside of the pars

*This increasing deafness can well be considered a masked sclerosis in those cases where there is a manifest sclerotic process in the other ear.

petrosa, e. g., in the ossicles, the external attic wall, and the walls of the external meatus Katz.[187]

These circumscribed foci consist:

1. Of pathologic new formed bone tissue, This has an osteoid character, is devoid of lamellated structure, has plump, irregular bone corpuscles, which are especially numerous at the places bordering on the normal bone, and possesses a different staining power from that of normal bone,

2. Of ostitic foci and abnormal medullary spaces (the spongifying process of Siebenmann). The inflammatory foci show, in fresh cases, new formed wide capillaries, on account of which Katz[181] identified this form of inflammation with the "vascular ostitis" of Volkmann.

The oldest changes are usually found in the region of the labyrinthine window, and there the origin of the disease should ordinarily be sought, according to Habermann[183] where the larger or smaller vessels pass from the periosteum into the bone.

That the disease may start in the ossicles is shown by a case of Katz[182] where we recongnize the usual anatomic substratum of the rarefying ostitis.

What is the causal relationship between the different pathologico-anatomic findings.

In recent cases we have only foci of ostitis vasculosa. We must seek the primary bony process, therefore, in this vascular inflammation. The new formed capillaries in the vascular, primary, inflammatory foci cause the bone to disappear and form the abnormal medullary spaces (foci of spongification.) This bony resorption is especially due to the osteoclasts, which Politzer[174] was the first to see in the pathologic medullary spaces.

The varied contents of these medullary spaces in the different stages of the disease leads to the belief that they are filled with new formed bone by the action of the oseoblasts Politzer[174].

That they may be filled, instead of with bone, with connective tissue, Politzer[174], or even with medullary substance Siebenmann[180], is to be regarded as the exception to the rule, according to the results of our previous anatomic investigations.

We see then, that the different anatomic findings in sclerosis, the circumscribed foci of new formed bone and the ostitic foci causing formation of abnormal medullary spaces are only different stages of one and the same process.

Whether changes in the sound perceiving apparatus follows the bone disease cannot yet be said with certainty. The rule is negative, according to the anatomic findings we possess.

As to the age, in which the affection is accustomed to appear, it ranges according to Ed. Hartmann, on the ground of 22 cases collected from the literature, between 20 and 50 years. That it can commence earlier is shown by a case of Scheibe[284], 8 1/2 year old deaf and dumb child, and the case of Katz[182], already cited, 1 year old child.

The history, even the most careful, gives no unimpeachable data as to when the disease began, because those affected pay attention to the affected ear and come to the physician only after the disease has existed a long time and a considerable part of the hearing is lost.

I have repeatedly found the condition accidentally in children of 10 to 14 years of age, who have come to the clinic on account of other ear affections, neuralgia, etc.

The index to the sclerosis of which the patient was ignorant—they thought they could hear normally—was usually the well known Schwartze's symptom of a hyperemia; occasionally very slight shining through a normal, delicate drum, which was free from all residues of previous inflammation.

The test for hearing showed that there was present an evident diminution of the normal power of hearing, of which the patient was ignorant.

Although this disease, on the side of the pathologic anatomy, has for the most part been made clear by the investigations of the last decade, the views of the different authors vary as to its cause, and actual nature.

I have already stated that less is to be expected from anatomic investigations than from careful, thorough histories which relate not only to the patient but also to his family. I demanded, in order to progress with the question of the etiology, that the family tree of otosclerotics be constructed

and the constitutional conditions of the individual members be very carefully examined.

Any one who knows the literature will agree with me that the histories reported often leave considerable to be desired.

Although the disease was considered by some authors to be a constitutional one, others especially lately Habermann[177] have taken the standpoint that it is a result of an otitis media, especially of an insidious otitis media following a chronic suppurative rhinitis.

But the more that the anatomic investigations showed that the main lesion was an entirely characteristic circumscribed focus, extending into the depths of the pars petrosa, whose distance from the mucous membrane of the middle ear spoke as much against the disease being a result of an otitis media as the fact that similar changes have hitherto not been found so deep in the pars petrosa as a result of otitis media, just so much more this view as to the origin of sclerosis loses ground.

In addition, in frequent cases of sclerosis, in spite of the most careful microscopic examination, no signs of a previous otitis media could be discovered, and the drum and middle ear at times seemed so normal that the assumption of a previous otitis media that had completely healed in the meantime had to be discarded.

While the fairly regular bilaterality of the process, although not always externally demonstrable, spoke in a certain way for the constitutional nature of the disease, the anatomic pictures, especially the histologic peculiarity of the new formed bone, gave the impression that the disease was specific.

Whether the disease is a syphilitic one, whether it is, as Gradenigo thought, a manifestation of syphilis hereditaria tarda (parasyphilitic affection of Fournier), for which many clinical facts speak, as well as the agreement in many respects with the syphilis of other cranial bones, must be undecided for the present.

Likewise, for the present the question of the influence of the other dyscrasiae mentioned by Katz must be left open.

Though it has been shown without question, that the bone process may come about without any previous otitis media,

some (Katz,) have returned to this as an etiologic factor, of course not in the earlier sense, but as the exciting cause which sets up the characteristic bony disease when the hereditary anomaly is present.

Therapy has not kept step with our advancing knowledge of the anatomy of sclerosis. Although the anatomic findings show the impossibility of fighting the disease with local mechanical means, we still find that physicians hold to those means with a tenacity that does not find its explanation in good results. We need only to refer to electric massage, which was abused in many cases.

I have never seen with this treatment a lasting functiona result, which corresponded to the labor and time expended, and cannot but believe that in cases where a real and lasting result was obtained there was an insidious exudative middle ear process, which was not discovered by the otoscope.

But that by this treatment there is frequently an amelioration of the tinnitus not etiologically explainable, I can no more deny than the fact that the patient after the treatment often feels subjectively much better.

These results, although palliative, justify the treatment. This must under all circumstances be a careful and guarded one, because as a local irritation, it can cause a rapid loss of hearing. The frequently unfavorable, irritative action of this and other local methods of treating the ear is easily understandable in sclerosis, since we know that the irritation of the mildest otitis media can awaken the slumbering bone affection.

In the year 1896, I[185] described my unsuccessful attempts at operative treatment and gave an exhaustive critique of this, especially the removal of the stapes, in which enthusiasts, ignorant of the anatomic nature of sclerosis thought they had an operation whose benefits would exceed that of cataract operation.

As to the internal treatment with phosphorus, of which Siebenmann said that it was beyond the stage of experimentation, the future must decide Perhaps in it we have a means which will arrest the disease in its very beginning; perhaps it would be good to give it prophylactically where there is hereditary pre-disposition.

At any rate, the pathologic anatomy of sclerosis shows beyond doubt that the internal treatment is the only way that deserves to be further tested.

Although according to our present views, this disease is incurable, we can often make the patient more comfortable. Where our therepy is to be applied, can be determined only after a careful examination of the whole body. And this can be done only by that aurist who is at the same time a learned general practitioner.

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XXIX.

CHRONIC NON-SPECIFIC LARYNGITIS; CLINICAL FORMS; TREATMENT.*

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General Remarks. Definition and Division of the Subject.

Chronic inflammation may attack any of the constituent parts of the larynx. Very rarely, however, are non-specific inflammatory processes of slow evolution observed in the deep layers of the laryngeal wall. On the other hand, they are quite frequent in the laryngeal mucosa. We will limit our study to inflammations of the mucosa, for which we will reserve the term "chronic laryngitis," without considering deep inflammations of the laryngeal walls—independent affections, to which it is properly the custom to devote separate descriptions.

In the group of chronic laryngitis thus delineated, the symptomatology is quite variable, and the laryngoscopic examination gives dissimilar results according to the cases. To make a profitable study of the affection it is necessary to attempt to group the cases of evident analogy. It is then recognized that the variations in appearance usually correspond to variations of seat, extent and histologic evolution, thus constituting distinct clinical forms, whose etiology, course and prognosis present well-marked differential characters and demand different treatment.

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*Synopsis of the report presented to the French Society of Laryngology, May, 1904. From the Annales des Mal. de l'Oreille, du Larynx, du Nez et du Pharynx, May, 1904.

A study of these clinical forms will be our present object. I shall group into one class the cases in which the dominant phenomenon appears to be an alteration of secretion, and separate them from a second class where either a diffuse or circumscribed hypertrophy presents itself clearly enough not to be confounded with simple inflammatory swelling, and therefore is of prime importance. I shall oppose, then, the "catarrhal laryngitis" to the "hypertrophic laryngitis." While the lesions require a grouping of their laryngitis into one of these forms, some patients have erosions or ulcerations of the mucosa. I shall discuss these, but without giving them, as heretofore, the name of "ulcerous varieties," and shall consider them as terminal complications of the form in the course of which they may be observed. On the other hand, I shall include in the group of hypertrophic laryngitis certain lesions whose inflammatory origin, in the majority of cases at least, is established, and whose description it seems to me ought to be separated from the artificial group of tumors. I also desire to speak of various hyperplasic productions, which it has previously been usage to consider as benign tumors, and describe them under the name of "polypi of the larynx."

We will commence our study by an exposition of tha knowledge acquired as to the histo-pathology of chronic laryngitis, as well as its general etiology. Then after taking cognizance of the laryngoscopic signs in different forms, we will study their special etiology, their symptoms, their course, their progress, their prognosis, and their diagnostic characters. We will finish with a discussion of treatment.

I.

Pathologic Anatomy, Etiology and General Pathogenesis of Chronic Laryngitis.

A—Histologic Lesions of the Laryngeal Mucosa in Chronic Diffuse Laryngitis. — Pachydermia Laryngis of Virchow.

In acute or sub-acute catarrhal laryngitis all the layers of the mucosa are not involved, except in severe cases. In

chronic laryngitis, on the contrary, all the layers are concerned, though to different degrees. The glands are constantly altered in varying degrees, often slight. They are almost always more or less hypertrophied. Along with the glandular hyperplasia, there is usually swelling of the adjacent connective tissue. The chorion is infiltrated with leucocytes. Later it is thickened by a new formation of connective elements, ending possibly in glandular atrophy.

The epithelium undergoes divers alterations. In the region where it is ciliated, it may be seen as a mass of cylindrical, ovoid, irregularly planted cells. In places it may be almost entirely desquamated. It may undergo another modification and become stratified pavement over a variable extent. At the same time there is observed an abnormal thickening of pavement epithelium at points where it normally exists, and to a marked degree; there may be a completely keratinized layer, containing eleidin.

On the surface of the chorion, the normal papillae are hypertrophied, and there are new-formed papillae at points where the pavement epithelium has replaced the normal ciliated. In extreme cases the swelling shows itself in the form of vegetating or non-vegetating tumors, especially in the interarytenoid region and upon the vocal cords. Virchow has given to this condition of the mucosa the name "pachydermia laryngis." The results of his researches and of other writers have been, in general, concordant, and establish the fact that laryngeal pachydermia constitutes a mode of reaction of the laryngeal mucosa to causes of inflammation of long duration, whatever they may be.

When lesions of this sort develop in the posterior portion of the larynx, the thickened epithelium has a tendency to desquamate in mass. The interarytenoid, polypoid products are finally detached spontaneously, whence the formation of superficial erosions, rhagades, or even of ulcerations proper, possibly increasing in depth and leading to perichondritis, chondritis, and cartilage necrosis. But these fissures are generally superficial, and heal rapidly if they are not aggravated by local inflammation.

When these ulcerations are seated at the anterior part of the larynx, they have no tendency to disappear, and the

process leads to a circumscribed hyperplasia. The lesions do not resemble those which Virchow described as diffuse pachydermia. However, this author has deemed it proper to apply the name "verrucous pachydermia" The contrast between the two, remarks Virchow, is more apparent than real, and depends upon the fact that in front the cartilages are more superficial and the vascular network more developed. But in both cases the productions are of inflammatory origin.

B—Histologic lesions of the laryngeal mucosa in certain circumscribed forms of hypertrophic laryngitis. Structure of polypi of the larynx.

Inflammatory nodules, singers' nodules or vocal nodules, have been studied histologically by numerous authors. The small nodules are simple epithelial thickenings, with or without keratinization, covering the normal or hypertrophied mucous lining. The more developed nodules may present two different types histologically. In the first type the body of the nodule is formed by a thickening of the mucosa composed of a very thin fibrous tissue, separated from the epithelium by a loose band of connective tissue. In the second type the body of the nodule is formed of a pseudo-myxomatous tissue traversed by veins and large dilated capilliaries with thin walls. Between this mass and the epithelium there is no lamellous zone—only a very thin base, surmounted by two or three layers of pavement cells. The reddish nodosities on the surface of the vocal cords, which often coincide with the nodules of "granular laryngitis" are only pachydermal swellings. It is to be seen, then, that the histologic study of "singers' nodules" permits the establishment of all degrees between simple pachydermia and fibrous or mucous polyps.

Fibrous polypi, which are very common, are almost always seated upon the vocal cords in the anterior region, in the vicinity of the commissure. Ordinarily single, the growth is rounded, sometimes of a lobulated appearance, and usually sessile. The dimensions vary from those of a grain of millet to a large cherry. Growth is slow. The tissue has the char-

acters of fibrous tissue. Their smooth, but unequal surface is invariably covered with stratified pavement epithelium, under which is found a layer of more or less developed papillae.

Eversion of the ventricle of Morgagni shows itself as a sessile, ventricular polyp, projecting into the larynx. It has long been regarded as a prolapse of the mucosa. But, outside of tuberculosis and syphilis, there may be observed ventricular eversions which are, it appears, genuine fibrous polyps of inflammatory origin.

Mucous polypi, which, like fibrous, are seated upon the vocal cords, are generally of small size. Like mucous polypi of the nose, they have wrongfully been given (Jacques) the name myxomata. The structure of mucous polypi of the larnyx differs from mucous polypi of the nose only in the absence of glands and glandular cysts. Renaut called them "pseudo-myxomata."

Papillomata of the larynx are either circumscribed or diffuse. Histologic examination invariably shows them to be composed of papillae made up of connective tissue and blood vessels, and covered with stratified pavement epithelum. When the papillae are short they are smooth. When they are long and uneven there is a villous type (rare) and a cauliflower type (common). Sometimes they are keratimized and eleidin is found (horny papilloma). Finally papillomata of the larnyx may be considered as hypertrophies in mass of papillae, and not as tumors.

Glandular hypertrophies, giving rise to extensive thickening of the mucosa in chronic catarrhal laryngitis, constitute, to speak correctly, small adenomata (Cornil and Ranvier). We also find in the larynx, but very rarely, circumscribed adenomata. The glandular culs-de-sac are here larger than normal, but their epithelial cells preserve their physiologic characters. They are sometimes surrounded by a fibrous shell, as I have observed in a private case.

Cysts of the larynx are almost invariably glandular cysts by retention. These cysts, rounded, usually gray and transparent, covered with a thinned mucosa and embroidered with vascular arborizations, have been rarely examined histologically. They are found in the form of a pouch full of liquid

mucus, either clear or opaque, or a viscid substance like honey. The internal surface is smooth and covered with a single layer of cylindrical epithelium.

Cavernous angioma of congenital origin is rare in the larynx. But more frequently there occurs in fibrous or papillary thickenings a sort of plaque, formed of anastomosing vascular dilations, with an aneurysmal or varicose appearance. There may also be observed upon the vocal cords ecchymous or real submucous hematomata, possibly giving rise to blood cysts.

The study of the histologic structure of these various circumscribed lesions leads us to group them with pachydermia laryngis, either in the typical anatomic form, or in other forms where the alterations predominate either in the epithelium or connective tissue (inflammatory nodules, granular laryngitis), or in the epithelium and hypertrophied papillae (papillomata), or in the connective layer of the chorion mucous or fibrous polypi. Finally, if we consider that the isolated glandular hyperplasia (adenomata), which may be observed upon the vocal cords, do not differ histologically from the glandular hypertrophies seen in other regions in the course of old chronic catarrhal laryngitis; that cysts of the larynx are always glandular; that true angiomata are rare in the larynx, where the lesions called "angiomata" are almost never anything more than vascular dilations associated with fibrous thickening or papillary hypertrophy; we arrive at the conclusion already indicated that the group of benign tumors of the laryngeal mucosa, clinically designated polypi of the larynx, is purely artificial and should be included with catarrhal laryngitis.

C—Etiology and General Pathogenesis of Chronic Laryngitis.

Rarely chronic at outset, chronic laryngitis, in a great number of cases follows acute or subacute inflammatory processes. Ordinarily, chronic catarrh installs itself only when the patient has previously suffered repeated acute or subacute attacks. After a number of recurrences, resolution is not complete; persistent lesions appear, and soon begin their slow, steady progresss.

Thus the affection develops in individuals who have coryza and chronic angina. In the genesis of laryngeal lesions, the dominant factor is insufficient nasal permeability, which obliges the patient to breathe through the mouth, and thus exposes the larnyx to direct action of air too cold, too dry or laden with dust and germs. However, outside of insufficient permeability, other affections of the nose (ozena) or chronic tonsillitis may induce chronic laryngitis by extension. Hypertrophy of the uvula, to which an undisputed etiologic value has long been attributed, is, in general, a consequence of chronic diffuse angina which so often coincides with laryngitis, and has no special influence upon the develop-men of the latter.

Chronic laryngitis has other intrinsic predisposing causes in the chronic non-specific affections of the lower respiratory passages, especially those which provoke frequent and prolonged coughing (chronic bronchitis, emphysema, etc.). In poorly compensated valvular lesions and progressive myocarditis, there exists a congestive state of the larynx which favors the appearance of the persistent lesions of chronic inflammation.

Chronic laryngitis is easily established in patients with chronic gastro-intestinal troubles. It is the same with migraine, rheumatism, hemorrhoids, neuro-arthritis. In the arthritic affection par excellence, fat diabetes, there is also chronic laryngitis in a number of cases. It is frequent in the gouty.

It is common to consider lymphatism as a state which specially predisposes to chronic inflammations of the prima viae. But this morbid temperament is often only the result of bad hygienic conditions which themselves create the predisposition.

These various predisposing causes are met with frequently in those subject to chronic professional laryngitis, where their profession exposes them to fatgue of the larynx and resulting local congestion (orators, preachers, comedians, singers, etc.). Functional overuse is one of the most potent exciting causes, especially at a time when the larynx is attacked by acute or subacute catarrh, however slight,

Another class of chronic laryngitis of professional origin

attacks individuals whose trade exposes them to continuous, prolonged, or repeated action of smoke or irritating dust (mechanics, chimney sweeps, stone masons, etc.).

Chronic laryngitis develops very often in heavy smokers. Alcohol in every form is more obnoxious to the larynx than tobacco, the use of which is often associated.

The influence of sex is only apparent. Men are not attacked more frequently than women, except as their manner of life exposes them more than women to exciting causes. Venereal excess, in many persons, has an undeniable reaction upon the vocal apparatus, and this applies to both sexes. In young persons onanism may provoke continuous laryngeal congestion. Utero-ovarian affections produce repeated congestion of the larynx, which may, in some cases, induce chronic laryngitis. The beginning of menstruation may cause congestion or aggravate a chronic laryngitis, or even induce submucous hemorrhages or hemoptysis. Pregnancy and the menopause may also give rise to phenomena of the same nature. Castration of the female appears only rarely to have harmful effects upon the voice.

The influence of age is not clear. The majority of patients are adults of 35 to 50 years. Exceptional in the old and in infancy, the malady is more common in childhood. In young boys it is frequently developed during the period of voice change. This precocious laryngitis disappears sometimes spontaneously at the change of voice or the approach of puberty.

In a broad way, chronic laryngitis is more frequent in cold and moist countries than in temperate and warm regions; but it is especially favored by sudden variations of temperature.

Is there a special etiology for polypi of the larynx? When we consult the writers upon this subject, it is easily apparent that there is none. The etiologic conditions are identically the same, in reality, as those of chronic laryngitis and we are fully justified, from this point of view, in considering them as products of inflammatory origin.

The process of repair in the laryngeal mucosa presents nothing special. When the causes exciting chronic laryngitis cease to act, spontaneous amelioration, then cure, may

supervene, more or less complete. But this fortunate course is not a necessary consequence to the development of in-inflammation. It may occur that the evolution of the various lesions is associated with diverse influences, the age of the patient, innate or acquired temperament, state of health—all the causes which may in different ways modify the vital properties of the tissues.

II.

A—Catarrhal Forms.

(*a*) *Simple Chronic Catarrhal Laryngitis.*

Laryngoscopic Appearance.—In recent and mild cases the lesions are little marked. The mucosa of the epiglottis and that of the entire vestibule shows a slight dull roughenening, most marked upon the ventricular bands. The membrane, a little swollen, has here a dull aspect, and appears moister then normal. The inferior vocal cords are often grooved with vessels. They have become grayish, roughened and moist and appear relaxed. In the laryngeal cavity there is mucus of variable appearance.

In other cases the vocal cords become rosy or even red to a varying extent. But the secretions are not more abundant than in the preceding condition. When the lesions have not led to interference with the nutrition of the muscles, the speaking voice and even the singing voice may be preserved. But in a great number of rather old cases, there is a paresis of the adductor muscles, and vocal tension is insufficient.

Finally, in the more severe cases, all these changes are present in highest degree in all parts of the laryux. Sometimes even, the presence of venous networks of varicose appearance, gives the larynx an aspect which Mackenzie called "laryngeal phlebectasia."

While the color and abundance of secretion are variable, thickening of the mucosa is rarely absent. The epiglottis may be deformed to the point of making examination difficult; the aryteno-epiglottic folds may be very much thickened. The ventricular bands may enlarge until they cover

the vocal cords. In some cases the tumefaction brings on dyspnea and wheezing.

The state of the vocal cords is not in constant relation to that of other parts of the larynx. They are sometimes roughened and swollen, sometimes grayish, flabby and relaxed. When the patient attempts phonation they vibrate unequally, and produce only raucous and broken sounds. This dysphonia is further increased by the swelling of the interarytenoid mucosa which hinders adduction of the vocal cords. In this degree the catarrhal laryngitis borders upon diffuse hypertrophic.

Symptoms.—General symptoms are completely wanting. The pain is nil, or only a vague sensation of intralaryngeal irritation. If the patient coughs, it is because of concomitant tracheitis or bronchitis. However, in certain cases, and under the influence of certain causes, there may ensue genuine slight attacks of laryngeal vertigo. The alteration of the voice is quite variable. The dysphonia may show every degree from slight roughening to deep hoarseness. Complete aphonia is rare.

Course and prognosis.—The course is steady, with more or less frequent exacerbations and ameliorations, consequent upon various influences. In children, cure easily follows when the causes cease to act. In the adult there is no tendency to spontaneous cure. The prognosis is never grave except as to the voice, complete re-establishment of this function being very difficult to obtain.

Diagnosis.—The laryngoscopic examination, which is indispensable, will suffice to differentiate simple chronic catarrh from syphilitic or tubercular laryngitis. But sometimes, to exclude tuberculosis with certainty, careful examination of the lungs and prolonged observation will be necessary.

(b) *Laryngitis Sicca.*

The etiology of this variety is somewhat special. It nearly always coincides with a fetid atrophic rhinitis, or with a chronic catarrh of the pharyngeal tonsil of the dry type. Laryngoscopic appearance—In mild cases, the mucosa is

rough, dry, sometimes dark. The vocal cords, which are gray or rosy, appear to be united at the anterior ends. The interarytenoid space is usually covered with greenish black mucus. The tension of the vocal cords is nearly always deficient. Sometimes shallow erosions or more or less deep fissures can be seen.

In older cases and during an exacerbation, there may be a more or less marked aggravation manifesting itself in an increase of secretion, with its persistent retention in the larynx.

Symptoms, course and prognosis—Hoarseness is rarely absent. It may be intermittent or constant. When there is an 'aggravation the dysphonia is accompanied by a raucous aggravating cough. Sometimes respiratory difficulties may set in, especially in neurotics, even suffocative attacks with glottic spasm. The cough results in the expulsion of hard concretions, sometimes followed by blood-streaked sputum. It is exceptional to see a real laryngeal hemorrhage—a laryngitis sicca complicated with the hemorrhagic.

Laryngitis sicca is one of the most rebellious forms of chroic laryngitis. The amelioration corresponds to that of the nasal or pharyngeal affection upon which it depends, but recurrence ensues in a majority of cases after a variable time.

Diagnosis.—The laryngoscopic signs of laryngitis sicca give it a characteristic individuality. The dyspneic complications may, in some cases, resemble those of laryngeal diphtheria, especially when accompanied by the expulsion of exudated membrane of uncertain character.

The diagnosis of the hemorrhagic form with true hemoptysis may give more serious difficulty, as hemorrhagic laryngitis has been observed in pulmonary tuberculosis.

B.—Hypertrophic Forms.

We shall distinguish only two classes of chronic hypertrophic laryngitis —diffuse and circumscribed—passing over the clinical study of laryngeal polypi. We will not speak here of the lesion described under the term chronic, hypertrophic, subglottic edema. It appears now to be well established that this affection is not autonomous and should be included either with rhinoscleroma or syphilis.

(a) *Diffuse Hypertrophic Laryngitis*

1. Late form.—Under this head I place those cases of old and severe chronic catarrhral laryngitis in which permanent and diffuse swelling of the mucosa takes lead over the secretory troubles. This form, which is rare, it seems to me, ought to be noted, but deserves no special description.

2. Pachydermic form.—In a number of cases, the dermopapillary transformation may show itself simultaneously over a great extent of the larynx. In some patients, the entire laryngeal cavity has a pale yellow tint, bright in some places, dull in others, and a dry appearance without trace of mucus; it might be said that the larnyx is covered with a skin-like membrane.

In some cases the lesion resembles absolutely the diffuse form of laryngeal leucoplakia, or milky plaques on the buccal surface—tobacco smoker's disease. As I have twice observed coincidence of these lesions upon the tongue and the larynx, I believe that we may call this curious lesion of the larynx laryngeal leucoplakia. Have we to do always in cases of this sort with syphilitic or parasyphilitic lesions, or simple pachydermic lesions? I am not in a position to answer this question.

Symptoms, course, prognosis and diagnosis of diffuse hypertrophic laryngitis.—The symptomatology, course and the prognosis of the first type, as well as the pachydermic type, present nothing special. The diagnosis of the generalized pachydermic form from the sclerous form of tuberculosis may in some cases necessitate bacteriologic examination and inoculation tests.

(b) *Circumscribed Hypertrophic Laryngitis.*

ANTERIOR CIRCUMSCRIBED HYPERTROPHIC LARYNGITIS.

(a) Nodular Laryngitis. Laryngoscopic signs.—Nodular laryngitis (singer's nodules) is characterized by the presence on the anterior third of the free border of the vocal cords, of two nodules, with dimensions varying from a small pinhead to a grain of millet. They are never pedicled. Sometimes

there is only one. Upon attempting phonation their summits are brought into juxtaposition, thus preventing complete approach of the vocal cords.

Symptoms and diagnosis.—When the nodules are very small, the spoken voice is, in general, good. The singing voice alone is affected. In some cases, especially when the nodules are of some size, there is observed a peculiar affection of the singing voice, diphonia, or simultaneous emission of two different sounds. More often than diphonia, is observed the phenomenon known as "coulage" of the voice. "The air escapes between the cords—the note is accompanied with a slight hissing" (Castex). When the nodules are somewhat large and there is acute catarrh, singing becomes impossible, and even the speaking voice is greatly roughened.

The diagnosis is obvious. The only thing of importance is not to mistake for nodules small masses of mucus at the point of election for these nodules.

Etiology and pathogenesis—course and prognosis.—In adults the influence of sex is marked (80 to 95 per cent. of women). In children it appears to be almost nil.

The influence of profession is very important. The great majority of patients are singers. Bad use of the voice, overuse of the voice—the same causes as for other forms of laryngitis in singers and orators, or rather the same cause, functional fatigue.

The prognosis is serious from a vocal point of view when the patient is abandoned to herself, and the most dilligent treatment is not always effective.

(b) Granular laryngitis.—I apply this name to a type of hypertropic laryngitis characterized by the presence on the anterior thirds of the vocal cords of protuberance resembling isolated granulations of the pharynx. Along with them the vocal nodules are generally more rough, larger and more rounded than the preceding form. This affection, often consecutive to the former, often develops quickly. It is an almost constant type in children, rare in men, extremely frequent in women. The symptoms are those of the preceding form, but not accentuated. The prognosis, grave in the adult, is relatively benign in children. Many cases cease after pub-

erty, either without treatment, or with a few precautions, hygienic rather than therapeutic.

(2) *Circumscribed posterior hypertrophic laryngitis (typical diffuse pachydermia of Virchow).*

Laryngoscopic signs.—This may show itself as a lesion almost entirely limited to the region of the vocal process, or, on the other hand, extending to the interarytenoid region.

(*a*) Apophysary pachydermia.—This variety is seated in the posterior part of the vocal cord. At a certain degree of development the lesion appears as an oval, bilateral tumefaction, occupying the internal border and the greater part of the superior surface of the vocal cord, with elevated margins and at its center a slight depression. The tumefaction begins at the internal side of the vocal process, and sometimes its development may be observed from the beginning.

(*b*) Interarytenoid pachydermia.—The lesions may confine themselves to this region and there remain a variable time but usually the hypertrophy extends to the posterior part of the larynx, the interarytenoid region, and invades it in the form of red or whitish protuberances or sharp, horny vegetations, villous and polypoid. These pachydermic masses prevent regular adduction of the cords and considerably increase the alteration of the voice.

Symptoms, course and prognosis.—The symptoms present nothing special. The progress of the affection is essentially chronic, and the prognosis from a functional point of view most grave.

Complications, ulcerations and so-called catarrhal erosions.—There is never really any extensive loss of substance, but some simple erosions in the front of the vocal apophysis take the form of fissures, or rhagades, in the interartenoid space.

Etiology and diagnosis.—Apophysary and interarytenoid hypertrophic laryngitis are affections peculiar to the adult man, especially drinkers and great smokers.

The diagnosis gives no serious difficulty. Sometimes, however, a bacteriologic examination is necessary and is not always sufficient to differentiate simple pachydermia from the symptomatic pachydermia of tuberculosis.

When only one of the cords shows lesions of pachydermic appearance, it is necessary to eliminate syphilitic gumma, epithelioma and circumscribed tuberculous infiltration.

3. Ventricular Eversion.

Other than in syphilis and tuberculosis, where it is relatively frequent, ventricular eversion may develop very rapidly after acute catarrhal laryngitis or appear in the course of chronic laryngitis. It most often develops at the expense of the inferior surface of the superior vocal cord, and probably also of the lateral surface of the ventricle. The eversion begins in the inferior part of the ventricle. Little by little the thickness of the ventricular band increases and the ventricular opening is replaced by a more or less red swelling, covering the greater part of the inferior cord. The symptomatology varies according to the volume of the swelling.

Diagnosis is usually easy. It is important to know that tuberculous laryngitis may begin with a superior corditis with ventricular eversion.

4. Horny papillary corditis.

I will describe under this name a rather rare affections as yet little known, which merits this designation because of its seat, structure and characters. From a histologic point of view, as Virchow has said, it is a form of pachydermia. It is characterized by a marked development of papillae covered with an extremely thick keratinized epithelium. The lesion has been seen under the glottis and upon a ventricular eversion, but it is almost constantly seated upon the inferior vocal cord, nearly always on one side. The cord is invaded either in part or in totality. Sometimes it is little changed, it shows only a dull, chalky pallor, like that of a "cat's tongue;" sometimes it becomes notched and deformed as if mangled. After being removed with the cutting forceps the tumor has a great tendency to recur. Up to the present the cases of definite cure are very rare. Both sexes are attacked; the majority of cases are in men of about 60 or older, but the affection has been seen in young girls.

III.

Treatment of Chronic Laryngitis.

A—General Considerations.

In general the first causal indication to be fulfilled, before even treating the larynx, is to do away with the lesions of the nasal fossa and of the epi- and naso-pharynx, if any exist, which is the rule. Exciting causes must be suppressed; the voice must be given absolute or relative rest and local irritation (tobacco smoke, dust, alcoholic liquor) avoided. In mild cases, causal treatment may suffice to produce cure. But usually direct treatment of the chronic laryngitis is necessary, and this is almost exclusively local, the really useful internal medicaments being few.

B—General internal medication.

We will only mention, in passing, certain medicines almost abandoned now, such as singer's syrup, etc. Bicarbonate of soda, chlorate of potash and tar are seldom used. Alkaline, sulphurous and arsenicated waters are rarely utilized to-day, except at watering places.

Medical treatment in the sub-acute attacks gives most service. Bicarbonate of soda in large dose (six to twelve grams per day) in cachets taken with a warm infusion of violets, or lemonade, sweetened with syrup of tolu, have marked favorable action. But the most useful drug in such cases, because of its elective, if not specific, action upon the congestive and catarrhal state of the air passages, is the benzoate of soda, an action which I first called attention to in 1885. But it must be understood that this drug acts well only in large doses (4 to 5 grams for children, 6 to 10 grams for adults). I often prescribe the following syrup, giving 3 to 5 dessertspoonfuls daily, at regular intervals, in a like number of small cups of infusion of violets or warm lemonade:

Benzoate of soda (neutral benzoin)		30.
Syrup of tolu		
Syrup de capillaire	aa	135

Warm and dissolve.

Quinin, antipyrin, or phenacetin, opiates (codein, peronin), may be used to advantage along with benzoate of soda.

In chronic laryngitis with paresis or fatigue of the laryngeal muscles I have found strychnin and nux vomica to be excellent. Some persons improve more under the latter possibly because of the brucin it contains independent of strychnin.

C—Thermal Medication.

The good effect of mineral waters, alkaline, arsenical and sulphuretted, in certain varieties of chronic laryngitis are almost solely due to their local action upon the mucosa of the air passages.

Our confreres at watering places have long since learned the great importance of employing the waters and they have special apparatus for inhalation, spraying, etc. I will limit myself here to an enumeration of the principal French sulphurous and arsenical waters whose action has long been favorably recognized. Of the first I would cite the Pyrenees, Bagnieres-de-Luchon, Eaux-Bonnes, Amelie les Bains. Saint Honore, Aix-en-Savoie, and Challes. Among the second, Mount Dore, Royat and Bourboule. Of those abroad I will mention only Schinznach and Ems, whose efficacy I have proved.

D—Local External Treatment; Massage.

External massage of the larynx. practiced in the Swedish method, appears to have been too much neglected by laryngologists. It requires various manipulations: effleurage, friction, vibration, etc. It probably has a decongestive action and promotes muscular contraction. Whatever its mode of action, it is certainly favorable in some cases.

Electrotherapy.—Electrization of the larynx with rheophores carried directly into it, or into the pharyngo-laryngeal recesses, is rarely utilized nowadays. Electrization of the anterior cervical region is, very properly, preferred.

Either galvanic or faradic currents are employed. The writers do not agree as to their relative efficacy in the cases with which we are now concerned.

For four years I have made use of sinusoidal currents (sinusoidal faradization) of moderate potential and frequency. These currents produce energetic but painless muscular contractions. They have thus far given me very encouraging results.

E—Local Internal Treatment.

1. *Inhalations of Steam and Liquid Sprays.*

For this purpose inhalers and atomizers are used. The inhaler should continuously produce a sufficient quantity of vapor to saturate the air going into the respiratory passages with each inspiration. A very practical apparatus, devised by Mandl in 1857, is the following:

A glass globe, a third filled with the liquid to be vaporized, heated by an alcohol lamp and furnished with two tubes, one communicating with the air outside, the other longer and bent obliquely. To the latter is attached a rubber tube with a large glass mouthpiece which the patient holds between the lips. A Russian coffee pot, with some modification, could serve as a useful and cheap inhaler.

The best drugs for inhalation are the balsams: balsam of Peru and benzoin, and certain oils (menthol, eucalyptus, oil of pine, etc). An alcoholic solution is prepared and added to water in the proportion of a teaspoonful to a glass of water. I often prescribe:

Tincture of eucalyptus,	
Tincture of benzoin, aa	60.
Oil of pine,	2.
Menthol,	4.

The oils of myrtle, sandalwood, citron and origanum may also be utilized. The latter, which is rather irritating, should be employed only in small doses and in especial cases.

Atomizers throwing vapor into the throat are the only ones to employ in the treatment of chronic laryngitis. I will not describe the well known apparatus of Siegle. The patient should place himself before the apparatus, his mouth wide open, and at the same time, with a towel in his hand, hold the tongue well out, and make deep inspiration.

For atomization, only aqueous solutions of substances soluble in water should be prescribed. Solutions of tannin, sulphate of copper, and sulphate of zinc, 1 to 5 per cent. are of service as astringents. As an antiseptic the solution of choice is a solution of pure carbolic acid in .25, .50 or 75 per cent. strength.

I refrain from describing the numerous apparatuses designed for the inhalation of vapor or atomized fluids; I wish to call attention, however, to one recently invented by Heryng (of Warsaw). I have seen it work, and it appears to be a step in advance.

2. *Instillation by Largngo-tracheal Injection.*

This method which is relatively recent and was first applied to the treatment of diseases of the bronchi and lungs, notably tuberculosis, is of service in the treatment of some sorts of chronic laryngitis. The instrument most to be recommended for instillation is the three ring syringe of Beehag, which has been excellently modified by Mendel.

Some patients can learn to make the injections themselves. but they are more valuable when done by the physician, under the control of a mirror. Mendel, who directs the jet either in the middle line of the posterior pharyngeal wall or into one of the glosso-epiglottic fossae, is easily able to make a part of the fluid enter the larynx, but some of it also goes into the esophagus, which is not always a matter of indifference. The preferable excipient for these injections is olive oil, sterilized, to which may be added menthol (1 to 3 per cent.) oil of eucalyptus (2 to 6 per cent.), etc.

3. *Topical Applications.*

These applications require the employment of an instrument appropriately called the porte-topique (applicator). Forceps, properly so-called, should be rejected because of their numerous inconveniences. It is preferable to employ a rigid steel cotton carrier, with a handle sufficiently long to be held like a pen, and having at its laryngeal extremity a screw with which to fix the tampon of absorbent cotton. Ac-

cording to the case, the application may be simply by contact or by friction.

The useful applications are either the astringent or mildly caustic solutions, or antiseptic solutions.

Among these, nitrate of silver is the most commonly employed, without fully justifying the confidence placed in it. I have long since substituted for it chlorid of zinc, weak solutions of which, 1 to 4 per cent., are most useful. Sulphate of zinc and tannin sometimes give better results than zinc chlorid. As a topical antiseptic I employ almost exclusively phenol sulphoricinate.

From 10 to 30 per cent. it answers all purposes. When it is badly borne, which is rare in the larynx, I have often advantageously used instead of it the sulphoricinated balsam of Peru.

Another application, which is astringent or caustic and also antiseptic, according to strength, is the following iodin solution:

Iodin	2.
Iodid of potassium	3.
Distilled water	20. to 25.

F—Endolaryngeal surgical operations.

Anesthesia of the larynx.—Only local anesthesia is safe for operations. To obtain it, freshly prepared, concentrated solutions of cocain should be used. Some add to this adrenalin. Lubet-Barbon gets slight anesthesia by insufflating equal parts of cocain and sugar, and complete anesthesia by instillation of a tenth or twentieth per cent. Moure is of the opinion that it is necessary, to complete the anesthesia by applying the agent with a cotton carrier. I prefer to commence this way. I employ, even in children of 12 to 15 years, strong solutions of cocain, 15, or better, 20 per cent. Some patients, however, have an idiosyncrasy, and this must be anticipated for fear of accidents. A good way to avoid these accidents in suspected persons, is to make them take 15 or 30 drops of Sydenham's tincture of laudanum in a little water. In persons whose pharyngeal reflexes are exaggerated and not aboli hed by the cocain, the operation should be put

off, and in the interval calm the hyperexcitability by means of large doses of bromid of potassium (4 or 5 grams daily) for four or five days. This method, despite the opinion of Chiari and Botey, may alone subdue some throats, which are exceptional, it is true.

Chemical and thermic cauterization.—The powerful and diffusible caustics (chromic acid, for example), should not be employed. Nitrate of silver in stick and very concentrated solutions of iodin are about the only chemical caustics of which I make use. It is very important to make the application with precision. I would say the same of the galvanocautery for which may be employed, according to the case, the point, flat blade or rounded cautery.

Scarification, curettage.—One may use for scarification the laryngeal bistoury, with or without protected blades. I have had constructed by Raoul Mathieu, laryngeal scarifiers with three parallel blades, which I have employed successfully for several years.

The laryngeal curettes of Heryng are to be recommended for the posterior and lateral subglottic walls of the larynx. For the upper surface of the cords, I have had Mathieu make small, rounded curettes, acting as a plane with a spherical surface.

Excision with the cutting forceps.—The tubular system of laryngeal forceps invented by Turck have some indisputable advantages and some inconvenience. I prefer the rigid, jointed forceps. Instead of the bulky Morrell Mackenzie forceps, the sliding forceps of Gottstein or Fraenkel are preferable.

In 1889 I had Aubry construct a cutting forceps with double joints, in two models, one of which opens antero-posteriorly, the other laterally. Other forceps to be recommended are those of Moure and Suarez de Mendoza. It is often advantageous following forceps operations to make topical applications, such as iodo-iodin or su'phoricinated phenol. The patient should keep his room for 24 hours and preserve silence for several days. Weak carbolic sprays may also be prescribed.

C—Treatment of various forms of chronic laryngitis.

1. Catarrhal forms.—Mild catarrhal forms call for the employment of prophylactic and hygienic methods. External massage, electricity, strychnin internally, and sprays may be used; in more severe forms touching up with weak solutions of zinc chlorid, sulphuretted waters in torpid forms, arseniated in congestive attacks. Grave catarrhal forms demand painting with strong solutions of zinc chlorid or sulphate scarification, etc.

The sulphuretted waters of the Pyrenees are often an effective adjunct in the treatment of laryngitis sicca. In subacute attacks with dyspneic symptoms steam gives the best results. In hemorrhagic attacks one may have recourse to astringent sprays (tannin, perchlorid of iron) and adrenalin.

2. Hypertrophic forms.—According to some writers nodular laryngitis should be susceptible of spontaneous cure by resting the voice, or even by changing the manner of singing (Holbrook Curtis) but in most cases surgical intervention appears preferable to me.

If the swelling is soft the application of a galvano-cautery may lead to its disappearance within a few days: but if the swelling is hard the cutting forceps is the instrument of choice. I have a pair of forceps made by Aubry which permit me, because of the one-sided bite, to do this operation with security.

The prognosis of nodular laryngitis should be guarded. Recurrence is not rare, and I have seen several such cases.

Nodular laryngitis and granular laryngitis of children may sometimes cure by rest and after puberty. But it is generally better not to wait. Applications of iodo-iodin, preceded or not by scarification or ablation with the cutting forceps are most efficacious. In the adult this treatment often acts marvelously, but the frequent recurrences darken the prognosis.

The treatment of posterior hypertrophic laryngitis is still more difficult, and little more can be expected than amelioration. The topical application for apophysary pachydermia appears to me to be sulphoricinated salicylic acid. The em-

ployment of the cutting forceps should be cautious in this region, because of the danger of laying bare the vocal apophysis which is here very superficial.

The lateral cutting forceps and the curette are, on the other hand, of great service in interarytenoid pachydermia. If the patient is suspected to be tuberculous, recourse may be had to sulphoricinated phenol, 30 per cent.

For eversion of the ventricle use may be made, according to the case, of the galvano-cautery point or the instrument of Mahu. Care must be taken of the underlying vocal cord.

If I had to treat a horny papiliary corditis I would resort to sulphoricinated salicylic acid and my antero-posterior cutting forceps.

PROCEEDINGS OF THE FRENCH SOCIETY OF OTOLOGY AND LARYNGOLOGY. ANNUAL CONGRESS.

Session of May 2, 1904.

The president, M. Castex, declared the session open and made the following remarks:

"My Dear Colleagues: You have honored me greatly, and I am very thankful to you for having designated me to preside over your work this year.

"If you are willing, we will conform to a pious usage and first honor our dead. Quite recently, the first of April, there died one of our former presidents, Camille Miot. Born at Moulins-Engelbert in 1838, Miot had been pupil and chief of the Blanchet clinic in the National Institution for Deaf-Mutes. His numerous works, which are fresh in your memory, revealed a surgical taste—operative procedures upon the tympanic cavity, diseases of the ear, foreign bodies in the nasal fossae, etc. Personally I had little acquaintance with him, but to the best of my information he merited the friendship of the illustrious, and I consider the following quotation applicable to him: 'Pitying the unfortunate, he lavished upon them his talent, rather than talents, giving to the poor that which came from riches.' I suggest that we give to Madam Miot and her family an expression of our condolences.

"We also have to mourn Dr. Latrille, of Poitiers, honorary member of our society.

"It should be a source of lively satisfaction to us to see our specialty increase year by year and conquer a new kingdom for itself. Does it not appear to you that we are colonials of whom the mother country may well be proud, for we have conquered vast territories hitherto unexplored?

"Our society may claim a good part of these results. It

has done much for the expansion of this specialty in France, much also for its dissemination by presenting its works in a clear and elegant form: a la francaise. What a pleasure it is to see the return with spring of the sessions which reunite us, specialists from abroad, from the provinces and from Paris. The work is nicely divided: anatomy, physiology, otitic and nasal affections, presentations of instruments, etc. The discussions are interesting, often inspiring, always useful.

"Permit your president to encourage in you that entente cordiale, so necessary to the encouragement of our work. We give more attention to the ideas of those who sympathize with us. Each of you here, having talent, may well offer the kindness of recognizing that of others: 'For the struggle which destroys lets us substitute union which multiplies.' This is the thought of a wise philosopher.

"Our work, moreover, is far from being ended. Will it ever be? How many questions are unsettled! I shall strive to direct this session for the best."

M. Joal, secretary-general, read his report.

M. Cadier, treasurer, elucidated the financial situation.

A commission, composed of MM. Moure, Jacques and Didsbury, examined the accounts of the treasurer.

Election of the bureau: Vice-president, M. Maurice Boulay, of Paris; Secretary of Session, M. Texier.

MM. Joal and Cadier were retained as secretary general and treasurer.

M. Ruault, of Paris, read a paper. (See page 326.)

Chronic Non-Specific Laryngitis. Clinical Forms. Treatment.

M. MENDEL.—I requested the privilege of discussing the remarkable report of M. Ruault, to defend the method of tracheal injection which he mentioned in speaking of the treatment of chronic laryngitis, and a method which I have introduced as a notable simplification of laryngologic technique.

At first let me say to you that it was not to laryngologists that I commended it; they are drilled in laryngeal

maneuvers and they easily perform injection without the mirror; I would say to them only: Employ injections of eucalyptolized oil, from one to five per cent.; give them daily for several weeks in tuberculosis, and you will obtain —without cough, without spasm, without reaction—functional and stethoscopic amelioration as surprising as those which I have observed for several years, and which Rosenberg and Beehag, the creators of this method, have remarked.

But I return to the technique itself, which is the subject under discussion. It constitutes a non-laryngologic method, so to speak, in that it does not require the employment of the laryngeal mirror. Permit me to explain it in a few words: It is based upon the fact that a moderate quantity of liquid thrown upon the posterior wall of the pharynx descends by its own weight into the air passages, gliding over the digestive orifice, which is always closed except in deglutition. Now to throw the liquid upon the posterior pharyngeal wall I have conceived two proceedings. One consists in projecting the liquid directly upon the wall. The other, more practical, consists in throwing it upon the lateral wall of the pharynx; from this point, because of the impulse given, the liquid continues with a whirling movement to the posterior pharyngeal wall. These facts are not imaginary; they may be established by the aid of the laryngeal mirror, and in fact have been by several laryngologic writers whose findings I have cited in a book which ought to appear shortly.

Let us now examine the objections of M. Ruault to this method. Here are his own words: "This method may indeed, in certain patients, cause a portion of the injected liquid to penetrate into the larynx, but it allows another portion to pass into the esophagus and stomach, which is not always a matter of indifference."

Now, how many observations does M. Ruault bring to the support of his assertion? Only one. That of a young woman 25 years old in whom he employed the simplified method. "A little of the liquid always passed into the stomach," says he, "and there resulted at the end of about a quarter of an hour, a rather painful sensation of burning

in the epigastrium, lasting and hour and a half or even two hours." As for myself, who have employed this method for five years, and made thousands of injections, I have observed this accident, but only as an exception, and an exception which I consider avoidable.

How is the liquid injected upon the posterior pharyngeal wall introduced into the digestive passages? Solely by a movement of deglutition. The digestive orifice is closed, and keeps itself closed, after the projection of the liquid, as is easily demonstrated. The pyriform sinuses are closed, as Lubet-Barbon noted in an examination which he made with me. Finally the occlusion of the digestive orifice may be shown in the cadaver; in six instances I performed tracheal injection according to my method and the greater part of the liquid was found in the air passages, a slight amount remained in the pharynx, and not a drop had passed into the esophagus. If the patient does not swallow at the moment of injection, and if he expectorates the excess of liquid which may remain in the pharynx after the injection, nothing will pass into his stomach. It is merely sufficient to instruct the patient, which is very easy. If the patient swallows a little liquid by inadvertence or negligence, he will feel a burning, as has been observed by M. Ruault, for it is an interesting fact that the air mucosa is much more tolerant to caustic solutions than the digestive mucosa.

Finally, the execution of the method, although it is easy, demands a certain skill which M. Ruault did not possess, but which he could easily acquire; he daily performs more difficult maneuvers.

It is none the less true that all the liquid injected does not always penetrate into the air passages, and a portion is lost. I have been reproached with this in other terms; they have criticized the imprecision of my method, but are they sure, in using the laryngologic methods, of injecting all the liquid into the air passages? Not at all, and a certain portion of the liquid is always, or almost always, rejected. Moreover, imprecision is constant in therapeutics, which is far from being an exact science. Its real criterion is, and always will be, the clinical result.

After answering the objections which have been made to

my method, allow me to state briefly its advantages. The two chief are these:

The proceeding is never disagreeable to the patient, for there is almost no instrumental contact, no mirror, no introduction of the laryngeal syringe. This advantage is not negligible and it is only necessary to compare the two procedures to be convinced. My daily injection is always perfectly borne; it is even demanded by patients.

Now, to how many patients is it possible to give daily laryngologic injections without nausea and repugnance? How often is it necessary to use cocain, which I now never use?

Another advantage: The simplified method does not require any special instruction; I have taught it at one sitting to a student, even to a nurse.

And if one desires to use it in the treatment of pulmonary tuberculosis he will find that the method can be better popularized than the laryngologic procedure.

M. VIOLLET—I wish in this connection to report the contradictory results of experiments recently conducted by M. M. Mendel, Lubet, Barbon and Viollet. There were three patients; two of them had been treated over a month, and the third had undergone many intralaryngeal injections. The liquid thrown upon the posterior pharyngeal wall of the patients thus habituated entered the larynx. It does not follow, however, that this would be the case in any patient, or those treated by unskilled hands.

M. RUAULT observed that the inconvenience of introducing fluid into the esophagus could be avoided in only one way—by carrying the medicament directly into the trachea.

M. JACQUES—Eulogy of the very remarkable report of M. Ruault is unnecessary; we all appreciate the value of his work which is just as thorough from the anatomic as the clinical point of view. I think we ought especially to congratulate him for showing the identity of benign tumors in general, and the absence of neoplasic specificity, a fact which is insufficiently set forth in the classic works.

ABSTRACTS FROM CURRENT OTOLOGIC, RHINOLOGIC AND LARYNGOLOGIC LITERATURE.

I.— EAR.

Midlde-Ear Disease in its Relation to Metastatic Abscess of the Liver and Other Viscera.

S. MacCuen Smith, Philadelphia. (*Archives of Otology*, Vol. XXXIII, No 2), reports 3 cases and refers to 3 other cases which he has personally observed of old chronic middle-ear suppuration in which acute exacerbations were followed by pyemia. In two instances the liver alone was involved, in two additional cases the lungs were also the site of multiple pus foci.

The most constant and characteristic symptom was that of severe pain in the right shoulder and neck, appearing synchronously with the beginning of hepatic suppuration.

Jaundice was a constant factor but it appeared so irregularly as to be of little diagnostic value, save confirmatory.

The history of secondary infection in each of these cases is sufficiently clear to warrant the conclusion that a metastatic abscess of the liver, or other viscera, may originate from a suppurative disease of the ear. *Campbell.*

A Contribution to the Pathological Anatomy of the Internal Ear and the Auditory Nerve.

Manasse, Strassburg. (*Archives of Otology*, Vol. XXXIII., No. 2). He reports the findings in seven cases of labyrinth disease complicating chronic middle ear diseases. In about half of the eases the extension of the inflammatory process from the tympanum to the internal ear was through the fenestra ovalis after destruction of the foot-plate of the stapes. Although necrosis of bone was noted in the simple cases of otorrhea, they were characterized more by a proliferation of tissue. In most of them there was an abundance of fibrous-tissue formation and the formation of

new bone. In the one tubercular case there was no such tendency, the newly formed fibrous tissue was scant and was mostly transformed into cheesy material. Habermann had previously made the observation that tubercular cases could be recognized by their great tendency to bone destruction.

Campbell.

Report of a Fatal Case of Chronic Suppurative Otitis with Cholesteatoma in the Drum Cavity and Antrum, Cellulitis of Almost the Entire Scalp, Thrombosis of the Transverse Sinus, Cerebral Abscess and Softening of the Cerebellum.

GUTTMAN, New York. (*Archives of Otology*, Vol. XXXIII, No. 3.) These conditions developed from an otitis media suppurativa chronica of eight years duration. The most important and most pathognomonic symptom in these cases is undoubtedly the pyemic fever. Next in importance was the choked disc in the right and the optic neuritis in the left eye. The extensive cellulitis of the entire scalp extending to the eyelids and face, was rather a disagreeable complication as it made the diagnosis more doubtful and interfered with treatment. The cellulitis was probably an extension of the edematous swelling which appeared posterior to the mastoid in the region where the emissary vein emerged. This, the so-called Griesinger symptom, together with the lack of swelling in the anterior part of the mastoid is a valuable sign of intracranial complication. Not much stress must be laid on headache as it is purely a subjective symptom. The slow pulse which made its appearance toward the end of the disease pointed strongly to the necessity of exposing the brain. In fact, so strongly did the symptoms of sinus phlebitis overshadow those of brain abscess, that without the symptom of slow pulse the presence of brain abscess could not have been determined.

Conservative expectant treatment is not justified where suppuration is progressing in these regions; surgical interference is imperative. Where there is evidence that septic material is being carried into the circulation the jugular is to be promptly ligated. The mode and extent of surgical interference must always be adapted to existing conditions. *Campbell.*

Report of a Fatal Case of Multiple Otitic Abscess in the Temporal Lobe, with Remarks.

JORDAN, New York. (*Archives of Otology*, Vol. XXXIII, No.3.) The patient, aged 39, had, from childhood, a purulent discharge from his left ear. 2 months before coming under observation he had pain in the ear with dull headache on the same side. He kept on working till about a week before entering the hospital when he complained of vertigo, drowsiness and his temperature reached 100°F. Eye examination negative.

Radical operation was undertaken, about 2 drams of pus esaped under pressure from the attic; attic and antrum were lined by the discolored necrotic granulation tissue. There was a defect in the tegmen tympani 12x7 mm. The dura was dark, did not pulsate or bulge into the opening. A carious erosion was found on the prominence of the semi circular canal.

The operation occasioned no improvement in his condition, the right side of his face lacked tonus, drowsiness increased, optic neuritis developed, slow cerebration and sensory aphasia became more marked, so 2 days later the wound was extended upward and backward and a narrow-bladed knife inintroduced into the temporal lobe in several directions. At the depth of 1 inch an abscess was found and pus to the amount of 1 ounce escaped. After enlarging the wound, the cavity was loosely packed with iodoform gauze. Next morning patient's condition was still unimproved; removal of the gauze liberated about 1 dram of pus, so a rubber tube was inserted. Four days later an abcess at a much greater depth was evacuated but stupor increased with total right hemiplegia, dilatation of right pupil, severe hiccough. By lumbar puncture 1 ounce of clear cerebro-spinal fluid was withdrawn with temporary good effect. It contained no corpuscular elements or bacilli. 48 hours later the temperature ran up to 103.4°F, he became deeply comatose and died.

Section through the left temporal lobe showed 4 distinct abscess cavities with disintegration of brain tissue extending into the lenticular, into the internal capsule and into the median part of the parietal lobe. The final collapse and

deep coma was due to an extensive hemorrhage into the pons and fourth ventricle. *Campbell.*

Operative Opening of the Mastoid Bone in Otitis Media Purulenta with Extension of the Disease Beneath the Mastoid Process.

LEIMER, Munich. (*Archives of Otology*, Vol. XXXIII, No. 3.) In 97 cases of acute middle suppuration with mastoid complications requiring operation 17 of these were cases of Bezold's mastoiditis. While ordinary mastoiditis most frequently occurs during the first three decades of life, Bezold's variety is found most frequently in the third, fourth and especially the fifth decades. The greater portion of the mastoid region usually remains free from swelling and tenderness while the inflammatory process shows itself below the oblique line of insertion of the muscles at the mastoid process and filling up more or less the retro-maxillary fossa.

Opening of the mastoid is begun by applying the chisel low down at the very tip of the mastoid process and separating the insertion of the attached muscles. The outer cortex is removed, the antrum exposed at its posterior end and then the rest of the mastoid process removed including the large cells which extend inward beyond the mastoid groove. The periosteal elevator is then pushed in above the soft parts underneath the base of the cranium in order to open any pus cavities there, and if there are accumulations of pus lower down in the neck a counter-opening should be made at the most dependent point *Campbell.*

II.—NOSE AND NASO-PHARYNX.

Nasal Fibroid.

WOOD, Nashville. (*Archives of Otology*, Vol XXXIII, No. 3.) A girl, aged 15, for two years has had nasal obstruction, frequent nose-bleeds and progressive facial deformity. In the early months pain about the face and forehead was so severe as to confine her to her bed for 3 months. It later subsided and has not since been a prominent symptom. For

6 months past the tumor has protruded from the left nostril in the form of a red from mass, one inch in diameter and disposed to bleed on manipulation. The separation of the eyes and flattening of the nose has produced typical frog-face. By the rhinoscope a red mass was seen protruding backward into the naso-pharynx.

On operation a preliminary tracheotomy was done and the pharynx tamponed. The external incision was made, exposing the left nostril and the tumor removed in two parts. The pedicle was attached to the under surface of the body of the sphenoid and the upper edge of the vomer over an area 2 in. horizontally and 1 in. vertically. The outer bony nasal wall, the lateral mass of the ethmoid and all the turbinals had disappeared. The tumor occupying the whole left nasal fosa and antrum of Highmore.

The patient reacted badly and died from surgical shock 22 hours after operation. *Campbell.*

Contribution to the Pathology and Treatment of Diseases of the Accessory Nasal Cavities.

ROEPKE, Solingen. (*Archives of Otology*, Vol. XXXIII, No. 1).

2. Three cases of empyema of the accessory sinuses. The caseation in all three cases was probably due to obstructed drainage After removing the diseased tissue recovery quietly followed

2. A case of pneumatocele of the frontal sinus brought about by occlusion of the naso-frontal duct, probably the remnant of an inflammatory infection of the right half of the nose.

3. A case of fetid purulent frontal sinus empyema with the production of crusts in a pronounced case of rhinitis. This case of atrophic rhinitis occurring on a scrofulous basis, aggravated by factory dust, had extended through a patulous naso-frontal duct to the frontal sinuses.

4. A case of fronta sinus suppuration following a pistol wound, and extraction of the bullet from the naso-frontal duct. *Campbell.*

Preparation and After Treatment in Nasal Operations.

H. BEAMAN DOUGLASS, New York. (*Post-Graduate*, May,

1904). Dr. Douglass thinks the same preparation should be made for nasal operations as for the operations in general surgery.

Cleanse the nose carefully externally, washing the integument of the nose and cheek with soap and water and thoroughly scrubbing. Gauze compresses may be placed over the moustache and held in position with strips of rubber plaster; cut away hair at the entrance to the nostril.

Three hours before the operation wash the interior of the nose first with peroxid of hydrogen 1-10 with spray, and then with weak saline solution. If much mucus or pus is in the nose, ten drops of tincture of green soap in a quart of water should be used as a douche, followed by weak alkaline solutions. The irrigating fluid should be 102 to 106 degrees and of proper density; a teaspoonful of salt to the quart will do this. It should be irrigated with little force and through the obstructed nostril.

Boil all instruments from 15 to 20 minutes. The hands of the operator are treated as in major operations. The operator and patient are gowned in sterile dress and all towels freshly sterilized.

Whenever adjusting head mirror, do it with the hand covered with a sterile towel.

Solutions used for anesthesia must be fresh and sterile. To anesthetize, spray first and then put in pledgets soaked in 4 or 5 per cent. cocain solution mixed with suprarenalin. A pledget of cotton soaked in 4 per cent. solution of cocain and placed under the upper lip against the spine of the superior maxillary bone intensifies the local anesthesia.

The best after results are obtained from the use of antiseptic powders and non-interfering plan of treatment.

Bleeding from the septal veins is the most frequent form of epistaxis. The vessels here bleed very freely if only slightly disturbed; and the hemorrhage is sometimes very prolonged. Pressure, peroxid of hydrogen, 1-1000 adrenalin, torsion, if artery can be grasped, electro-cautery, are the methods to employ.

After tonsillotomy wait fifteen minutes for the cessation of hemorrhage, during which time the patient gargles with a solution of peroxid of hydrogen. If the artery is spurting

from the center of the tonsil, catch the bleeding point and twist the artery. This is the only method really worth using in cases of arterial hemorrhage. If the hemorrhage is not arterial, and persists after fifteen or twenty minutes cauterize with the electro-cautery or else sew the cut surfaces of the tonsil together. If the hemorrhage continues over half an hour the patient generally faints from loss of blood and this serves to check the hemorrhage.

Richards.

Empyema of Frontal Sinus, Followed by Extradural Abscess and Abscess of Frontal Lobe. Operation and Death from Hypostatic Congestion of the Lungs.

PAUL S. MERTINS, Montgomery, Ala. (*The American Journal of Medical Sciences*, April, 1904.) The patient was a boiler-maker, 40 years of age, and had suffered for six months from violent headaches steadily increasing in severity until a swelling the size of a goose egg appeared in the region of the glabella. It was white, fluctuating, very tender, and had a hard rim with thickened periosteum. There was a history of syphilis sixteen years ago, and two years previously a blow from a hammer on the top of the head from which he was unconscious for several days. The abscess was incised and one ounce of foul, dark colored pus was evacuated. This was followed by complete relief of pain which returned the third day. When the head was held forward the flow of pus increased and the pain became less. Vomiting followed a few days later and there were three convulsions.

When first seen by the author, patient was in bed suffering greatly. Cerebration was slow but relevant; no paralytic phenomena; pupils small, equal size, and reacted well; patellar reflexes good; hand grasp strong; no rigors. The anterior end of the middle turbinate was greatly swollen. Temp. 97.5; pulse 78; respiration 20.

On probing the wound necrosed bone was found, and in several places the probe seemed to enter the cranial cavity. Diagnosis of extradural abscess was made, and on the following day a three-inch incision was made in the integument of the forehead, through the seat of the abscess. The pericranium had been dissected from the bone by the abscess,

and on its being drawn back by retractors, an area of necrosed bone the size of a silver dollar was exposed. It was a dark brown color and presented the appearance of a pepper-box top from the numerous fistulae from which pus oozed at each respiration. A half-inch trephine was applied to the center of the necrosed area, and on the removal of the button, an extradural abscess containing about one ounce of pus was found. The dura was adherent to the frontal bone all around, and formed the bed of an abscess. Removing the necrosed bone opened the left frontal sinus, and about one-half ounce of pus was found in it. The right frontal sinus was opened and found to be healthy. The dura appeared to be very thick; was of a dirty, yellowish color and pulsated only on the left side. No fistulae could be found. A hollow needle was introduced on both sides with negative result on the left side, but with the finding of four or five drops of pus on the right as the needle was withdrawn. The wound was irrigated and iodoform gauze dressing applied. The next eight days the wound was dressed daily, urine frequently passed involuntarily; great irritability with semistupor; transient paresis of the right internal rectus, and optic neuritis on the right side. The right frontal lobe was then aspirated as the patient seemed to be in more than usually stupid condition. On introducing the aspirating needle in the right frontal lobe the depth of three-quarters of an inch it was felt to enter a cavity and pus flowed into the aspirator. The opening was enlarged and one and one-half ounces of creamy pus and a yellowish lump of broken-down brain tissue were evacuated. Iodoform gauze drain was inserted. There was immediate improvement in the patient's condition.

There was steady improvement for the next six days, the abscess cavity growing smaller and the discharge less. At this time there was a marked change in the condition with hypostatic congestion of the inferior lobe of the right lung, and coughing up of blood. Three days later the left lung became involved and all the conditions much worse. Death occurred two days later from exhaustion following the pulmonary conditions rather than directly from the abscess.

At the autopsy a depressed fracture the size of a hammer-head, about one-half inch deep was found on the left tem

poral bone near the sagittal suture. The bone underneath was healthy. On removing the calvarium, the dura was found adherent around the original trephine opening, both to the bone and to the brain. The membrane appeared normal. The brain appeared normal except at the point of operation where an opening 1 cm. in diam. led into the abscess cavity. The abscess was entirely walled off by a thick lining membrane, and the brain tissue about the abscess was in a nearly normal condition. The inferior lobes of both lungs were in a condition of hypostatic congestion and in the right lung there was a cavity the size of a walnut filled with clotted blood.

The author regards the abscessas the direct result of the empyema of the frontal sinus, the posterior wall of the sinus being necrosed and a fistulous tract leading from the sinus to extradural abscess. *Richards.*

Treatment of the Suppurative Diseases of the Accessory Sinuses of the Nose.

E. Fletcher Ingals, Chicago, Illinois. (*Illinois Medical Journal*, 1904.) For suppuration of the antrum Ingals prefers in the majority of cases Hunter's method, that is, the removing of the last bicuspid or first or second molar, or the opening made through the space left by a tooth already lost.

A conical bone drill 6 mm. in diameter or a diamond point drill-trephine of the same size is used, and under cocain this operation is nearly painless. A gold tube can be fastened with clamps to the adjoining teeth. He prefers, however, rubber tubes 6 mm. in diameter, 19 to 35 mm. in length, and with a calibre of 4 mm.

The cavity is kept clean by washing it out with watery solutions of 50 per cent. hydrogen peroxid or boric acid, and injecting once a day a 5 per cent. solution protargol or 20 per cent. argyrol or one to 5 per cent. solution of zinc sulphate. If this treatment does not succeed within three or four months, the advantages of a radical operation through the canine fossa should be urged upon the patient.

The operation with immediate closure of the canine fossa wound is preferred. It is especially important in curetting

the roof of the cavity not to injure the infraorbital nerve, and also that all angles and recesses be cleared of all diseased tissue. After the curetting is over, the cavity is swabbed with 95 per cent. carbolic acid and lightly packed with iodoform gauze.

Acute inflammation of the ethmoidal cells is treated with a spray containing two grains of cocain, 1/10 grain of suprarena in, eight grains of boric acid to the ounce of water, thoroughly applied several times daily. With chronic cases, all obstructions are to be removed and free drainage obtained as far as possible. In operating upon the ethmoid cells great care should be exercised not to break through the cribriform plate.

Affections of the sphenoidal sinus are probably very generally overlooked as the normal opening of this sinus is so high up that it is impossible to employ treatment effectively through it. and in the majority of cases it is not practicable to inspect the normal opening of this cavity until the posterior end of the middle turbinate has been removed. This being done, the anterior wall of the sinus may readily be inspected if the nasal cavity is of normal size. The anterior wall of the sinus may be opened by hook or forceps, trocar, sharp spoon, punch forceps or trephine. The trephine makes the smoothest opening and is to be preferred. Cure may be expected in from two to four months.

In suppuration of the frontal sinus Ingals uses a guarded pilot burr which is used from within the nose. The pilot is a flexible steel wire, and may be used straight or bent in any direction, and long enough so that it will project 5 mm. beyond the end of the burr when the burr has been slid over it. He also has a longer pilot which will project 9 mm. beyond the burr, so as to prevent any possibility of its working down from the frontal sinus when the canal is being enlarged. Before using this burr in the anterior part of the nose there should be thorough removal of any obstruction in the naris. He has been able to make a good sized opening into the frontal sinus through which gauze can be packed. The patient is anesthetized with chloride of ethyl, the operation requiring but a few seconds, four to six.

For the radical external operation he recommends that the

incision extend from the junction of the middle and inner third of the supraorbital margin in a curved line forward and downward to the root of the nose, about 6 mm. above the level of the internal canthus. The periosteum being divided, the opening is then made with the chisel and mallet; the cavity thoroughly cleansed of all disease; the wound swabbed with 95 per cent. carbolic acid; the naso-frontal canal enlarged by a Krause trocar guided by the little finger introduced into the nostril. The original incision is entirely closed, a gauze strip left in the wound coming out into the nose. The external wound is then closed, partly or completely, and an antiseptic dressing applied. If there is no fever and the dressing appears sweet, the packing may be left in the sinus for six or seven days, being then removed through the nose.

The more radical operation of Killian is briefly described. Recurrence of suppuration sometimes takes place and may necessitate a second or third operation. This statement probably refers to the results of the Caldwell-Luc operation, as after operations in which the sinus is completely obliterated it is doubtful if recurrences occur. *Richards.*

The Removal of Septal Spurs and the Correction of Deviations of the Septum by More Rational Methods.

CHARLES NELSON SPRATT, Minneapolis, Minn. (*American Medicine*, May 7, 1904.)

Resection of the Nasal Septum for Correction of Deflections.

WILLIS S. ANDERSON, Detroit, Mich. (*The Physician and Surgeon*, March, 1904.) The methods advised are practically the same as those of Freer and White, in the papers of which the operation is discussed more in detail. Spratt thinks Freer's elaborate instrumentarium unnecessary, and himself uses the chisel for the most part. He thinks that a Fraenkel-Swift speculum, a Sajous knife, a small elevator for the flap, a thin chisel, a pair of Heymann cutting forceps and plenty of cotton swabs are all the instruments that are necessary.

He holds the flap in position by a spatula, and packs the

nose lightly with iodoform gauze for twenty-four hours, replacing this then by fresh gauze, and finding after forty-eight hours that the flap is quite firmly attached to the septum and subsequent packing is unnecessary.

Anderson packs both nostrils with strips of lint impregnated with subnitrate of bismuth, packing the formerly obstructed side firmly and the free side loosely. Perforations occurred in some of his earlier cases but he thinks that after thorough familiarity with the technique of the resection operation, the perforation should occur but rarely.

Richards.

Salpingoscopy.

Joseph C. Beck, Chicago, Illinois. (*Medical Journal*, 1904.) The salpingoscope is an instrument for examining the region of the Eustachian tube and naso-pharynx, and consists of a tube with an ocular piece at the proximal end and a prism mounted by a light with a small curve at the distal end. Electricity is the source of illumination and is turned on or off by a switch on tbe handle of the instrument. It is introduced the same as the Eustachian catheter, being thoroughly cleansed and the prism covered with lysol or soap so as to prevent steam from covering it like a laryngeal mirror.

The eye is placed to the eye-piece and contact made by turning on the switch, then by moving the instrument gently forward and back, the outline of the ostium tubis with its anterior and posterior lips, is seen. As one is looking through a prism, the image is an inverted one, while the parts situated close to the prism appear very large, and those farther away, extremely small. Both openings of the tubes can be seen through one nostril.

The author has used it in a great variety of conditions and has found that it is an aid in diagnosis worth applying; that it requires considerable time to familiarize oneself with the technique and to learn the comparative sizes of the structures, and that its use is particularly of the value in the naso-pharynx in examination of the ostium tuba.

The salpingoscope was introduced by Valentine, whose

article may be found in the Arch. f. Laryn., 1903, Bd. XIII, Heft. 3. *Richards.*

III.—MOUTH AND PHARYNX.

The Submerged Tonsil.

THOMAS J. HARRIS, New York. (*Medical News*, July 9, 1904.) The submerged tonsil, even though hypertrophic and well below normal may give rise to a not insignificant group of symptoms such as peritonsillar abscess, non-suppurative tonsillar inflammation, pain in the form of sore throat, constant collection of mucus in the larynx, difficulty in using the voice, and occasionally, in the case of extensive adhesions, certain ear symptoms, the most common of which is earache. It is also a causal factor of considerable value in chronic catarrhal otitis. Since the attachment of the muscles known as the anterior and posterior pillars are in close relation to the pharyngeal mouth of the Eustachian tube, and as in the case of adhesion of these pillars and the tonsil all normal movement is restricted or lost, pressure of such a tonsil must of necessity interfere somewhat with the proper blood supply of the Eustachian tube.

Cough may be a most annoying sympton without expectoration; may have existed for months and may defy all internal medication. There is often an associated dyspepsia, with coated tongue and inactive bowels. The writer thinks that the average tonsillotomy is an incomplete operation, as most physicians have been in the habit of removing any excessive tissue which is present and then desisting. The case then passes out of sight, but in after years returns complaining of quinsy or some of the other symptoms enumerated or diphtheria. As a result, the lay or professional man has been led to the conclusion that tonsillotomy was without avail.

The treatment is essentially surgical. All medication is of no avail. The tonsil should be freed from its adherent tonsillar pillar, and if necessary, from the posterior pillar, and removed in whole or in part. The freeing from the pillar

is an indispensable step in the operation. The plica triangularis will often have to be cut loose in order to free the gland.

The author uses cocain, first painting with 10 per cent. solution and then injecting 10 to 15 m. of one-half to one per cent. solution. He prefers the tonsil punch to any of the other instrument although the tonsil can be removed with a variety of instruments. He has had no experience with the electro-cautery method of Pynchon. The chief concern is to avoid cutting the tonsil pillar. With the punch there is but little hemorrhage.

Only one tonsil is operated on the same day, and for the pain and discomfort which follows, orthoform in the form of an emulsion or five grain tablet is used. The other tonsil is removed one week later. *Richards.*

Lingua Nigra.

JOSEPH C. BECK, Chicago, Illinois. (*Medical Journal*, 1904.) The patient was 34 years old with a history of muscular rheumatism for several years; complained of a peculiar scratchy feeling in his throat near the back of the tongue, about one year's duration, worse in the recumbent posture. On examining his throat he noticed on the back of his tongue a dark mass which he attempted to wash off, and not succeeding, pulled off in pieces with a spoon, but it soon returned in the same locality.

On examination there was found a dark mass of a dirty brownish color, broadest at the base of the tongue at the circumvallate glands, gradually disappearing down to a point at the anterior third of the tongue. On removal the mass was found to be composed of a number of filamentous hairs, dark in color, having all the appearance of hornified epithelium which did not take stain well. The color of hairs was due to the aged condition of the epithelium, and its hornification, a constant increase of the normally present pigment. It is a combination of hyperkeratosis and pigmentary growths.

The local application of a 10 per cent. alcoholic solution of salicylic acid or 10 per cent. solution bichloride of mercury

was the treatment, together with the curette. Strong solutions of peroxide of hydrogen may also be used.

Richards.

Hemorrhage After Tonsillotomy; Its General Consideration.

H. JARECKY, New York. (*Medical Record*, April 30, 1904.) After the eighteenth year hyperplastic changes take place in the tonsil that has been the seat of repeated inflammations, causing the interstitial fibrous tissue to increase, and preventing the retraction of the blood vessels. This explains why hemorrhages rarely occur in children. Vessels which are cut slantingly are more apt to bleed than where they are cut squarely across.

The removed tonsil should be examined to see whether it shows the presence of any large artery which may require immediate treatment.

The wounding of the anterior pillars can always be avoided by separating them from the tonsil by means of a blunt hook. Bleeding from wounding the pillars is slight but usually persistent. It can be controlled with the galvanocautery.

Ulceration is usually the cause of secondary hemorrhages. After operation it is important that nothing be taken that will scratch the wounded surface or that is any way difficult of digestion.

Fibrous tonsils ought to be removed with the cold wire snare.

Cases of bleeding have been reported after all methods of operation. The author reports two cases of secondary hemorrhage, due probably to an injury to the plexus of veins at the base of the tonsil. If the bleeding does not cease spontaneously in a few minutes after the operation measures should be taken to a stop it. A hypodermic of morphia often acts like a charm. If one or two vessels cause the trouble they should be seized with forceps or tenaculum and twisted, while if the bleeding is from the whole surface of the tonsil or venous oozing, the Paquelin or galvanocautery should be applied. In persistent uncontrollable hemorrhage two ligatures can be passed from the posterior pillars through the anterior ones, and then tied so that the tonsil is folded on

itself or a recess formed in which gauze can be packed. There is little danger of injuring the internal carotid artery with the ordinary tonsillotome.

In case of bleeding use a good light and clean the parts to ascertain the cause; anesthetize or use morphine to quiet the patient; and never operate without being prepared to cope with hemorrhage. *Richards.*

IV.—LARYNX.

Further Experiences with Intratracheal Medication.

P. S. DONNELLAN, Philadelphia. (*American Medicine*, May 14, 1904.) Donnellan's first report is dated December 18, 1901. Since that time he has found intratracheal medication to be of great value in cases of pulmonary tuberculosis inasmuch as medicines can be introduced through the trachea without disordering the stomach and with quick absorption. In suitable cases the expectoration is diminished, the heavy masses of muco-pus being replaced by frothy mucus in which the tubercle bacilli are less numerous and are occasionally absent.

The injections are given daily at first and then at longer intervals. Pure olive oil, distilled glycerine and any of the fluid petroleum oils make good menstrua. Creasote, menthol, guaiacol, camphor, codein, and iodoform are used in accordance with the amount of cough and the individual symptoms.

He uses a specially devised syringe of glass with a metal ip. The reviewer has used this syringe to some extent. While it answers the purpose very well, it is readily breakable at the point where the syringe screws into the metal laryngeal tip. A syringe with a metal barrel and an asbestos plunger would seem to answer the purpose better and be quite as easily sterilized.

Dr. Donnellan reports a number of cases showing benefit from the injections. *Richards.*

The Larynx in Beginning Pulmonary Tuberculosis.

W. G. B. HARLAND, Philadelphia, (*American Medicine*, June 25, 1904), reports some cases showing the practical value of an examination of the larynx in beginning tuberculosis of the lungs, and thinks that in all cases of suspected lung trouble, even when the evidence on physical examination is insufficient, that an examination of the larynx should also be made, as this examination may give the first clue to the presence of the iung infection. Should the larynx be the chief seat of the disease, the diagnosis and prognosis must be founded on the local appearances present.

The diagnostic import of the lesions observed in the larynx will depend somewhat upon the findings found after a careful study of the case as a whole. The larynx changes observed early in tuberculosis of the lungs may be congestive or anemic. In the congestive cases there is apparently a somewhat persistent sub-acute laryngitis with reddening of one or both cords; due partly to cough and partly to infection of the deeper under lying tissues of the mucosa by tubercle bacilli. Such changes are more frequent when the tuberculosis infection has followed grippe or a series of colds. In the anemic cases there is a general pallor of the mucosa of the larynx. The tongue is covered with a whitish coating, indicative of gastro-intestinal disorder, and the veins at the base are swollen. The vocal cords may appear congested or may be very pale. The tissues are often pale from edema.

The well-known more characteristic lesions of the disease appear later. *Richards.*

V.—MISCELLANEOUS.

Yohimbin; Its Use in the Treatment of Eye, Ear, Nose and Throat Diseases.

J. H. CLAIBORNE, New York, and EDWARD B. COBURN, New York. (*Medical News*, July 9, 1904.) So far as the ear, nose and throat are concerned, Dr. Coburn has ascer-

tained that a two per cent. solution of yohimbin appreciably benumbs the mucous membrane in two minutes; produces marked anesthesia in five minutes and the maximum degeee of anesthesia in ten minutes. This anesthesia lasts from one-half to three-quarters of an hour, sensation then gradually returning, and being completely restored in one and one-half hours. It affects the mucous surface only.

On being applied to the turbinals in the nose there was at first temporary swelling with considerable hyperemia, followed by some contraction of the tissues. On the tonsils, uvula and faucial pillars little change was noted except slight flushing. There was at first salivation, followed by dryness, but no puckering or constricted feeling as after the use of cocain.

Mixed with equal parts of 1-1000 adrenalin chlorid and applied to the turbinals there was considerable contraction of the tissues, not so marked as with adrenalin alone but much more so than with the yohimbin solution alone. As compared with cocain, the pallor, anesthesia and contraction of the tissues is not nearly so pronounced. Used for nasal operations the hemorrhage was more marked than when cocain was used, and the sensation not as thoroughly obtunded.

Secondary hemorrhage did not occur.

Cauterization of the tonsils to which yohimbin had been applied was rather painful.

Its advantages are that it is non-toxic; the anesthesia is of long duration, and it does not markedly contract the tissues; its taste is only slightly bitter, and it does not cause complete contraction of the throat and mouth.

Its disadvantages are that it does not keep well; it does not contract the tissues; there is some hyperemia and hemorrhage after operation, and it produces salivation.

Richards.

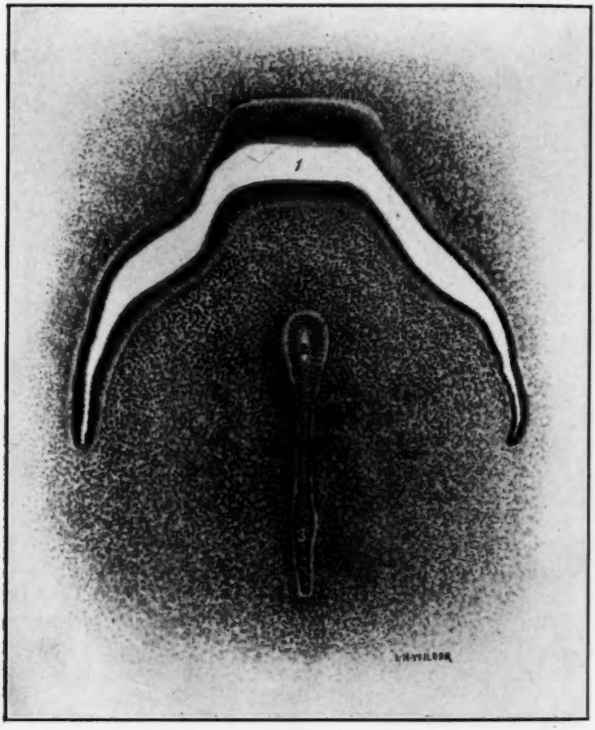

FIGURE I.

Transverse section through the larynx of an embryo pig 11 m m. long.

1. Cavum pharyngis.
2. Dorsal space, which is occluded above by the proliferation of the epithelium of the interarytenoid incisure; below it is continuous with the tracheal lumen.
3. Concresced epithelium.

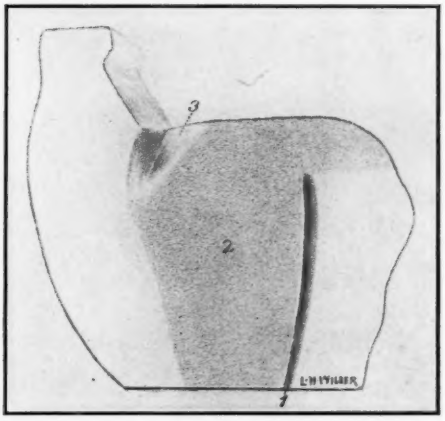

FIGURE II.

Reconstruction in wax of the larynx of an embryo pig 14 m m. long, enlarged 66 2-3 times. The reconstruction has been divided in the sagittal plane to show the area of concrescence.

1. Dorsal space communicating with the tracheal lumen below. The cephalic extremity is occluded by the concrescence.
2. The shaded area shows the extent of the concrescence.
3. Ventral space (aditus laryngis).

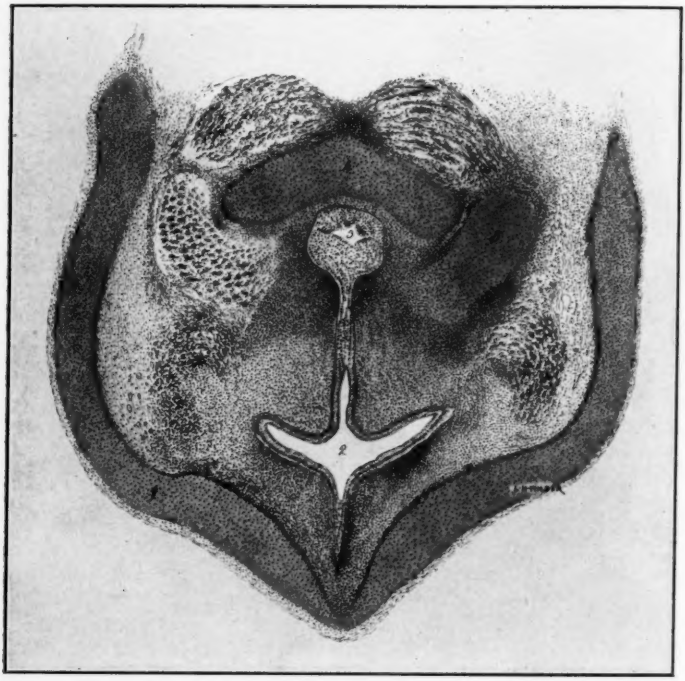

FIGURE III.

Transverse section through the larynx of an embryo pig 40 m m. long.

1. Thyroid cartilage.
2. Ventral space, which by the separation of the concresced epithelial surfaces is extending dorsalward.
3. Dorsal space.
4. Cricoid cartilage.
5. Lower part of the arytenoid cartilage.

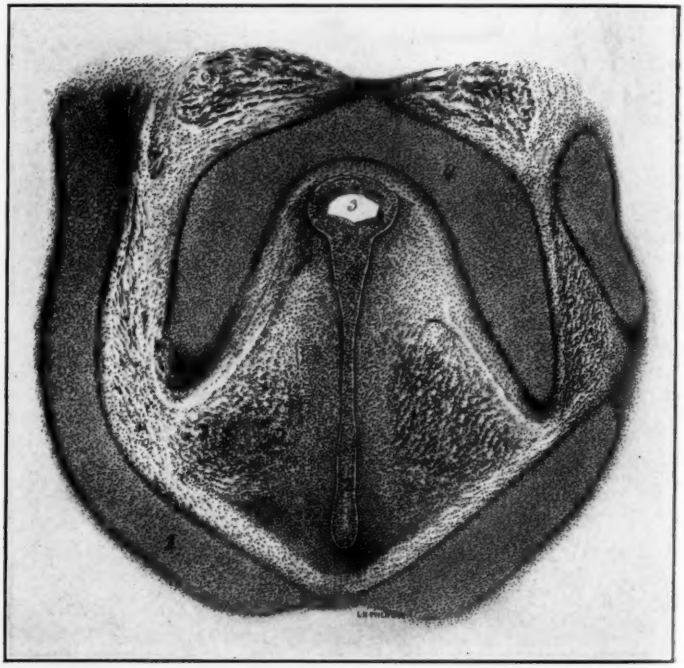

FIGURE IV.

Transverse section through the larynx of an embryo pig 45 m m. long, below the plica vocalis.

1. Thyroid cartilage.
2. Concresced epithelium (still intact).
3. Dorsal space.
4. Cricoid cartilage.